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ON GLAUCOMA.

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THE question as to the cause of glaucoma has interested ophthalmologists for half a century and has been the subject of zealous study and numerous investigations. An incredible amount of labor, diligence, and intelligence has been devoted to the solution of this question, and still to-day we have not yet learned in what the essence of glaucoma consists.

While the symptomatology of glaucoma has been studied in a most exhaustive way, and thereby many points brought out of value in respect to the doctrine of glaucoma, the pathological investigation of this affection has left much to be desired. The latter fact is due in part to the relatively small amount of available material and in part to the last stages of the disease in the eyes that are examined, when it is no longer possible to say which changes are primary and which have developed in the slow course of the disease. When we add that many authors have tried to reconcile the pathological conditions found with some preconceived idea of the nature of glaucoma, it is evident that many of these pathological investigations have little value. This is particularly true of many investigations of an early period. In recent times the scope of the investigations has been broader, and equal attention has been paid to all parts of the eye, whereby our knowledge of the pathology of glaucoma has been greatly extended.

Physiological experiments also have been undertaken in

great number without leading to a definite conclusion, although important facts with reference to the circulation of liquids in the eye have been discovered.

If we now ask why it is that, notwithstanding the expenditure of so much labor upon the study of the nature of glaucoma, we have to-day so little definite knowledge of the true character of the affection, it may be explained as being due to the fact that we have started with a false conception, since we have wished to find the essence of glaucoma in the increased tension, and since we have hoped that with the discovery of the cause of the increased tension we would at the same time discover the actual causes of glaucoma.

This conception of the importance of increased tension goes back to v. Graefe, who expressed his position in the matter in the following words: "The semiotic conception of glaucoma rests upon the increase in intraocular tension with its effect upon the functions of the optic nerve or retina."

This conception runs through all the modern theories of glaucoma like a red thread; wherever the cause may be sought, whether in the narrowing of Fontana's spaces, or in an inflammation of the choroid, or in a morbid process in the optic nerve, it is always the effects of the cause of the disease upon the lymph circulation of the eye and the resultant increased tension which are the actual chief factors.

This course of thought is justified when we are dealing with the so-called inflammatory glaucoma which begins with a marked increase in tension; in simple glaucoma, on the contrary, particularly in those in which there is little or no increase in tension, it is not justified. And yet it seems that all authors, with but a few exceptions, such, for example, as Mauthner, have considered chiefly the increased tension and have derived all the other symptoms from it, without devoting the requisite attention to simple glaucoma in which this course of thought is not justified.

It may be proper here to discuss the relation of simple to inflammatory glaucoma since in the course of time different views have been held, and indeed in recent times the view has been held by Schweigger, de Wecker, Knies, and others,

that simple glaucoma differs from inflammatory glaucoma and is to be regarded as a separate disease. It is well known that v. Graefe in his first publication expressed the opinion that simple glaucoma did not belong in the same class with true glaucoma, but that it was to be regarded as an affection of the optic nerve with excavation; this view, however, he gave up later. The more the cases were collected and the knowledge of glaucoma increased, the more the opinion grew that simple glaucoma belonged in the same category with true glaucoma, and that the cause of the disease was the same in the two. And in fact we see these two forms of glaucoma that are so different in their clinical appearance change their form and pass from one variety to the other. We see how an acute inflammatory glaucoma may lose its inflammatory character and assume the aspect of a simple glaucoma, and on the other hand we see a simple glaucoma suddenly become inflammatory. This is the case not only with simple glaucoma having increased tension, but also of the forms in which there is no increase in tension. It is particularly this latter form that one has wished to cast out of the category of glaucoma, although in its entire clinical course it resembles simple glaucoma and presents every sign of it except increased tension. Thus persons have wished to make glaucoma and increased tension identical, and deny that glaucoma existed without increased tension. Every unprejudiced ophthalmologist must concede, however, that this conception is false. I need not give statistics of such cases here for they are not rare; but I shall cite one case that I observed almost daily some years ago, and I am convinced that every experienced ophthalmologist will recall one or more cases of this sort in his practice.

An old man, a high ecclesiastic, visited me in the autumn of 1889 with the complaint that his vision had diminished recently and that in particular he was bothered by a "cloud" which lay over the lower part of the field of vision; he had also for some time had difficulty in going about at dusk or when the light was bad. There was no pain and no other complaint. On examination I found V = $\frac{5}{6}$; field of vision normal except for a defect in the lower part that extended upward nearly to the

point of fixation. The light sense was reduced (4.5*mm* by Förster's photometer). Tension normal. Fundus normal. A diagnosis of glaucoma simplex in each eye was made. An iridectomy was done in each, and strychnia was injected. Nevertheless the diminution of vision went on steadily but slowly; the field became more and more contracted; and the optic nerve became atrophic and excavated. In this manner the disease progressed for years and ended in complete blindness and marked excavation of the disc without there being ever a trace of increased tension. Neither I nor the patient who followed the changes in his eyes with painful attention could ever discover any increase in tension. A few days before his death an attack of typical inflammatory glaucoma appeared in the right eye and soon after in the left.

There can be no doubt that in this case the cause of the disease, of four years' activity, was of a glaucomatous nature, although it produced merely diminution of vision and atrophy and excavation of the nerve; and only a few days before death was their increase in tension accompanied by the characteristic circulatory disturbances of inflammatory glaucoma.

The increase in tension is thus only something accidental —a result of the glaucomatous process, just as the functional disturbance and the excavation are results of this process; it cannot be regarded as an essential element and much less as the cause of the glaucoma. The cause is therefore the same in simple glaucoma as in inflammatory, the difference being that in simple glaucoma the nervous elements of the eye are chiefly affected and early functional disturbances result, while in inflammatory glaucoma the cause acts upon the circulation in a disturbing way and brings about increased tension besides other results. *Hence increased tension and glaucoma are not identical.*

I am not the first to express this idea, indeed it is generally known; yet it seems to be forgotten or overlooked whenever a new theory of glaucoma is constructed.

A second point of fundamental importance for the doctrine of glaucoma is that simple and inflammatory glaucoma are really one and the same disease and that the same morbid process is active in both cases. With whatever symptoms

glaucoma may begin, whether with the most severe objective and subjective signs or without evident objective disturbances and with such slight subjective annoyances that the patient scarcely takes note of them, it is still one and the same disease which appears with varying symptoms that frequently characterize only different stages of the same morbid process.

Let us see now how these fundamental ideas accord with the theories of glaucoma held at present. It is not my intention to present and discuss all the theories that have been advanced, but I shall limit myself mostly to the modern theory of retention which has been so popular in the last twenty-five years.

Common to all the retention theories is the supposition that the cause of the glaucoma lies in an interference with the outflow of liquids from the eye either in a blocking of Fontana's spaces (Knies, Weber, and others), or in a blocking of the channels at the posterior pole of the eye (Stilling), or in both these factors combined (Laqueur). When the channels of exit are blocked, the liquid accumulates, the tension rises, and glaucoma appears. The increased tension is the cause of all the disturbances in the eye which characterize inflammatory glaucoma. The marked disturbance in circulation (the inflammation?), the shallow anterior chamber, the diminished vision, the excavation of the nerve head, etc.—all are but the results of the increased tension.

In regard to the closure of the channels of exit, and particularly of Fontana's spaces, various opinions have been held. Knies (*Arch. f. Augenh.*, vii.), on the ground of pathological investigations, assumed that an *indurating inflammation* at the sclero-corneal margin was the primary trouble, and that this led to a blocking of Fontana's spaces, as well as an adhesion between the root of the iris and the cornea. But although pathological examination usually revealed such an adhesion at the root of the iris, it was not correct to regard this adhesion as the cause of the glaucoma, since probably, on the contrary, it is to be regarded as a result of the glaucomatous process. It should be remarked that these investigations were in old cases in which for one reason or

another enucleation had been done. In the few cases in which by chance an eye with fresh glaucoma has been examined (*e. g.*, Birnbacher, "Ein Beitrag zur Anatomie des Glaucoma acut.," Gratz, 1890), this adhesion was wanting. And from a clinical point of view we must acknowledge that an inflammatory process of the sclero-corneal junction as a cause of acute glaucoma has never been observed. In the numberless cases of acute glaucoma that I have seen develop, as I may say, under my eyes, there was never any evidence of such an inflammatory process. On the contrary, the attack has appeared like lightning from a clear sky in an eye until then apparently healthy. The Knies supposition of an inflammatory process at the sclero-corneal junction as the preceding cause of glaucoma seems therefore more than doubtful. The cellular infiltration which Knies, Birnbacher, and others have found in Fontana's spaces, and in the tissues about Schlemm's canal, need not be regarded as the expression of an inflammation. The excessive pigment which is frequently found in this region and doubtless comes from the uvea, indicates that these cells and this pigment have been carried here by the currents of liquid and from some cause have been deposited in the narrow canal and block it. I shall return to this point later.

Weber, indeed, does not consider an inflammatory process to be the cause of the blocking, but he believes the blocking to be due to a venous stasis in the ciliary body which swells and presses the iris against the cornea. But whether the blocking of the channels of exit depends upon an inflammatory process or upon a narrowing of the channel, the cause of the increased tension and of the glaucoma is, according to the theory of retention, the prevention of the free outflow of liquids.

This theory may be applicable in cases of inflammatory glaucoma in which, as I shall show, retention in fact plays an important rôle, but it is not applicable in cases of simple glaucoma. Here the increased tension plays a subordinate rôle. It often exists, sometimes in high degree, but as a rule in simple glaucoma the tension is not greatly increased, and in many cases there may be no increase at all. The

latter cases are of the more importance in the explanation of glaucoma, because they show that glaucoma may exist without tension and without retention. Since simple and inflammatory glaucoma must be regarded as being due to the same morbid process, as I have shown above, it follows logically that retention is not the cause of glaucoma. Therefore another common cause of both varieties must exist. We may seek for this along various directions. We may start with the increased tension, we may start with the excavation of the nerve head, or best, we may start with the functional disturbances which appear in simple glaucoma without there being increased tension.

Simple glaucoma is chiefly characterized by the absence of all objective symptoms. There are no circulatory disturbances in the anterior half of the ball, little or no tension, and the excavation appears, as a rule, as a late sign. Externally and in its interior the eye appears normal in every respect, and only its functions are disturbed so as to indicate that a severe morbid process is under way.

This functional disturbance consists in a diminution of central vision, a narrowing of the field of vision, and a diminution of the light sense. In different persons these disturbances appear in different degrees and in varying reciprocal relations, so that there is no fixed rule in regard to their appearance. The central acuteness of vision often tends to diminish considerably while peripheric vision is still good; in other cases the contrary is the case, and in others the two disturbances advance with equal rapidity. Not infrequently the contraction is to the nasal side, and this special form has been regarded as the type of contraction in glaucoma. However, there may be contraction to other sides, and even sector-like defects are so frequent that we cannot speak of a typical glaucomatous field. The disturbances of peripheric and central vision thus appear in various forms, the field sometimes suggesting that of atrophy of the optic nerve and sometimes a hemiopic defect. The condition of central vision and of the field of vision, therefore, gives us no clue in regard to the nature and origin of the disease, since the disturbances might be attributed as well to an atrophy

of the optic nerve as to a morbid process in the retina. But the deduction of the light sense in simple glaucoma is a true indication of the nature of the fundamental cause. This symptom of glaucoma is generally overlooked or but lightly regarded. After Förster first noticed it, Mauthner was the first to call attention to its great importance in interpreting the nature of glaucoma. Mauthner came to the conclusion that the reduction of the light sense was one of the most frequent, not to say permanent, symptoms of glaucoma, and from my own experience I can fully confirm Mauthner's observations. In the beginning of simple glaucoma it is not infrequently the hemeralopia which is the only prominent symptom and leads the patient to the physician. The central vision and field are normal or but slightly affected, the tension is often normal, and only the reduction in the light sense calls the patient's attention to his affection. This hemeralopia may appear several years before the other symptoms of glaucoma. Such a case is the following now under my care:

An old lady came to me in the fall of 1892 complaining that she had difficulty in going about after dusk, and that reading or fine work was difficult even by daylight, since the eyes soon tired. On examination it was found that she had a low degree of myopic astigmatism with V = $\frac{6}{9}$, field and fundus normal. Light sense reduced = 4*mm* Förster's photometer. Not being clear in my mind as to the cause of this disturbance of vision, I endeavored to improve it by correction of the refractive error, tonic treatment, and subcutaneous injections of strychnine. The condition became better. In 1898 the patient noticed that her vision had grown worse in both eyes, but particularly the right, in which from time to time she felt a slight but lasting pain. It now appeared that in both eyes a simple glaucoma had developed with diminution of vision, contraction of the field, and beginning excavation of the nerve head. The right eye was the more affected, and in this the tension was manifestly increased. In the left eye tension was normal. Iridectomy in both eyes and 10–15 strychnine injections repeated three times a year have kept the vision as it was in 1898.

The hemeralopia thus appeared five years before the other

symptoms of simple glaucoma became manifest, and there is no doubt that the hemeralopia was the first evidence of the beginning glaucomatous process.

However, the hemeralopic disturbances are not always noticed by glaucoma patients, because the reduction of light sense is so slight that it is overlooked while it is confined to the periphery of the field. I believe that there are very few, if any, cases of simple glaucoma in which careful examination of the periphery of the field will fail to show a reduction of the light sense. This reduction, whether affecting the entire field or only a part of it, is one of the most characteristic, and certainly the earliest, of all the symptoms of simple glaucoma. Since hemeralopia must be considered the first expression of the glaucomatous process, we must ascertain wherein the cause of the hemeralopia lies.

According to all experience, the cause of hemeralopia is a functional disturbance of the rod-and-cone layer of the retina. In consequence of imperfect nutrition, these elements fail to functionate in a normal manner. They require a stronger excitation in order to permit perception. While ordinary daylight is still sufficient to allow these elements to functionate, the lesser light of dusk does not permit perception. Vision at dusk is completely lost or greatly reduced. The cause of the subnutrition of the rods and cones is due to a disturbance in the source of nutrition for these elements. Since the layer of rods and cones is not provided with vessels, and the retinal vessels nourish the inner layers of the retina only, we must seek the cause of the subnutrition in the inner layers of the choroid, whose vessels supply the outer layers of the retina. In the choroid, then, we must seek for the cause of simple glaucoma.

What the nature of the process may be is *a priori* hard to say, since pathological investigations on this point are incomplete and relatively rare. However, it has been shown that an extensive atrophy of the choroid is a constant condition in glaucoma. Goldzieher, as early as 1875, spoke in favor of the view that a choroidal atrophy was the cause of glaucoma. Fuchs also held this view, and thought that in most cases one could discover changes in the choroidal

pigment with the ophthalmoscope, although these changes were frequently limited to the anterior portion of the fundus and thus escaped notice.

For my part, I agree entirely with the opinion expressed by Goldzieher and Fuchs. I have often been surprised to find fundus changes in simple glaucoma that were in every respect similar to those occurring in retinitis pigmentosa. I believe that the cases of acquired retinitis pigmentosa with glaucoma following, which we find reported in the literature, are nothing but cases of simple glaucoma with marked atrophy of the choroidal pigment. In other cases of simple glaucoma these changes are slight and are limited to an irregular distribution of the pigment, giving the fundus a mottled appearance, or they may be altogether wanting. This, however, does not exclude the existence of an atrophic process, since in so marked a choroidal process as retinitis pigmentosa not infrequently all signs of atrophy of the pigment are wanting.

We can imagine an atrophic process taking place in the chorio-capillaris leading to a disturbance of nutrition in the rods and cones, without, in the beginning at least, injuring the pigment. In the later course of the disease, differing in different cases, greater or less changes appear in the pigment, and cases of red simple glaucoma are rarely found in which there is not more or less atrophy of the pigment, sometimes over the entire fundus, sometimes more particularly in the periphery, and again in a zone about the disc.

The functional disturbances in all the varying forms of simple glaucoma are in perfect accord with the supposition of a choroidal atrophy of this sort. The diminution of the light sense, the narrowing of the field of vision, and the diminution of the central vision may readily be regarded as results of the atrophic process. The atrophy does not extend at once over the entire choroid, but begins usually in the periphery and gradually extends toward the centre, although sometimes the reverse of this process is observed. Limited areas extending from the periphery toward the centre and islets of atrophy may be present and cause the different defects in the field which we see in simple glaucoma.

Passing from the functional disturbances to the second principal symptom of simple glaucoma, the excavation of the nerve head, let us try to discover how far this symptom is the result of the choroidal process. Most authors since v. Graefe have regarded this symptom as the direct result of increased intraocular tension, as the expression "pressure excavation" indicates. I shall hope, however, to show in what follows that this idea is not correct.

The intraocular tension exercises its influence uniformly over the entire ball, distending its walls in every direction. The resistance of the walls offers an obstacle to the distension, and the two forces are opposed.

What the conditions would be if the intraocular tension increased while the resistance of the walls remained normal is hard to say, since we have no clinical experience on this point. The bulging of a portion of the wall with increased tension, as in leucoma adherens with secondary glaucoma, gives us no information in this regard, for here there has been disease in the eye and the resistance is weakened. Locker's physiological experiments on normal eyes have shown that the intraocular tension must be raised to 135*mm* Hg in order to produce excavation of the disc. Since this pressure is much higher than is ever found in glaucoma, where it ranges from 30*mm* to 80*mm* Hg, and only exceptionally reaches 100*mm*, it is probable that the increase in intraocular tension, as we find it in diseased conditions of the eye, is not sufficient to distend the walls of the eye, if they are healthy and of normal resisting power.

The matter is different when the resistance is diminished by a preceding inflammatory process such as a sclero-choroiditis, for then the normal tension may cause a bulging of the affected part, as in intercalary staphyloma and the like. The conditions are similar with regard to the nerve head.

The normal disc possesses sufficient resistance to withstand not only the normal tension, but also a considerable increase. In cases of adherent leucoma, in which the tension is often considerably increased, we find not infrequently that the disc is entirely normal, although it has been exposed for years to the action of increased tension. Even in

glaucoma with greatly increased tension we find at times no excavation of the nerve head. Recently I saw a case of chronic inflammatory glaucoma with greatly increased tension of six months' duration, in which the nerve head was not excavated or even atrophic in appearance, although V = 0. Many cases like this have been reported. Even Mauthner described such cases. An increased tension need not cause an excavation of the nerve head, but on the other hand it seems that an excavation can take place without an increase in tension. In numberless cases of simple glaucoma with tension normal or but slightly increased, an excavation is found. I recall a case of simple glaucoma in a young woman, in which there was no increase in tension, but yet the nerve head was excavated to a degree that I have never seen equalled. The excavation thus may come on independently of increased tension, and therefore should not be designated as pressure excavation. The excavation must depend upon the fact that the tissues of the disc have lost in greater or less degree their resistance. Only two elements can come into consideration—the nerve fibres and the connective tissue composing the lamina cribrosa. One can scarcely believe that the nerve fibres offer any great resistance to the intraocular tension, and in genuine optic-nerve atrophy with complete disappearance of the nerve fibres the nerve head shows no signs of excavation. The slight depression left by the disappearance of the nerve mass cannot be designated as an excavation and has nothing in common with the glaucomatous excavation.

It is therefore the connective-tissue lamina cribrosa which furnishes the resistance against the intraocular tension. The lamina cribrosa is formed of numerous interlacing bundles of connective tissue, leaving openings for the passage of the nerve fibres. Although the sclera furnishes the main portion of the lamina cribrosa, the choroid takes some part in its composition. A great number of vessel-bearing tracts from the surrounding choroid pass into the nerve trunk and branch in the anterior layers of the lamina cribrosa. These vessels have a thick structureless elastica and a well-developed muscular sheath. The tracts themselves are so

richly pervaded with elastic fibres that these in fact make up the principal part of the connective-tissue structure of the lamina.

Pathological investigations have shown that in glaucomatous excavations a considerable atrophy of this connective-tissue supporting framework is always found. Thus, for example, Czermak and Birnbacher (*Graefe's Archiv*, xxxii., 4) found in a relatively fresh case of glaucoma the lamina cribrosa pushed back 0.65*mm* behind the plane of the lamina vitrea of the choroid. The tracts of the lamina cribrosa were forced together from before backwards and the lamina thus considerably thinned. Only the fibres from the posterior part of the sclera were clearly to be seen, the anterior scleral and the choroidal fibres not being clearly made out. "The papillary tissue is atrophic." Only in fresh cases are capillary loops to be found in the posterior layers of the lamina cribrosa, while in other cases they are wholly absent. An atrophy of the elastic tissue of the lamina — namely, of the inner portion which arises from the choroid — is unmistakable.

Whether this atrophy is the result or the cause of glaucomatous excavation is difficult to decide from a pathological standpoint, since the investigations made have been on eyes with old glaucoma in which it was no longer possible to distinguish primary from secondary changes, and only the examination of a fresh simple case could give us definite information. Since, however, as we have seen, the cause of the excavation is to be sought not in the increased tension but in a diminished resistance of the papillary tissues, it is probable that the atrophy is primary and the excavation secondary. As the anterior portion of the lamina is a direct continuation of the choroid, we may assume that an atrophic process running its course in the latter would also involve the elastic tissue and thus lead to the excavation.

The atrophic process in the choroid is to be regarded as the cause of the functional disturbances occurring in simple glaucoma. The diminution in the light sense is a result of this process and the malnutrition of the rod-and-cone layer which it causes. The contraction and other defects in

the field, as well as the diminution of central vision, are results of the increasing malnutrition and the gradual degeneration of the rods and cones. Since this atrophy of the choroid, as a rule, extends to the elastic fibres of the lamina cribrosa, the latter lose their power of resistance and the intraocular tension causes an excavation. The excavation of the disc is indeed one of the most constant and most characteristic symptoms of simple glaucoma and is independent of the existence or non-existence of increased tension.

Increased tension is frequent in simple glaucoma and is the chief feature of inflammatory glaucoma. I have thought that it might be explained in the following way as a result of the choroidal atrophy: The intraocular tension is dependent upon two factors—the resistance and elasticity of the surrounding membranes and the pressure exerted upon them by the contents of the eyeball. The first factor is of little importance, but the pressure of the included liquids is of great importance. When the liquid increases, the tension increases, and *vice versa*. The quantity of the liquid contained in the eye is regulated chiefly by two factors—secretion and absorption, which as long as the circulatory relations are normal balance each other and keep the tension uniform. With either of these factors disturbed, the circulation of the ocular lymph is altered and the quantity of liquid is increased or diminished until secretion and absorption balance and the tension adapts itself to the new conditions.

There is a third factor, however, which plays no small rôle in regulating the circulation of liquids in the eye, although it has hitherto been disregarded. I refer to the pressure which the choroid exerts upon the liquid contents of the ball and particularly upon the vitreous. It is evident that the tension of the choroid is considerable, as is shown by Donders's observation that when all the coats of the eye are divided together the choroid retracts from the margin of the wound and leaves the sclera exposed. This contractility of the choroid is doubtless due to the numerous muscle fibres which accompany the larger vessels throughout the entire membrane. Besides, the choroid gains considerable contractility through Bruecke's muscle which acts as a tensor.

But, besides the function of the choroid as a restrainer of the intraocular tension, it has the further function of increasing the lymph circulation of the eye. If we think of the choroid as surrounded by a muscular network and suppose that this network contracts, the result will be that the entire choroid is drawn together, thus exercising pressure upon the vitreous and increasing the currents of liquids in the eye.

An atrophic process in the choroid, when it involves the outer portion, the layer of large vessels, exerts a paralyzing effect upon the muscle network and diminishes the contractility of the choroid, and thus causes a slowing of the lymph current. So long as secretion and absorption remain undisturbed and balanced, the intraocular tension remains unchanged. But in consequence of the slow circulation the ocular liquids pass more slowly through the channels of exit, and all the formed elements of the blood, such as cells, fragments of cells, pigment granules, and the like, are deposited in the channels, gradually blocking them. The liquid in the eye increases and the tension goes up. But with the increased tension the canals are forced open and the circulation becomes normal until the canals are blocked again and the increased tension returns (prodromal stage).

It may happen that the stoppage of the channels of exit is so considerable that the increased tension is not sufficient to open them. The tension then increases more and more and disturbances in the circulation of the blood are added. These disturbances are exclusively of venous character and depend upon a hindered exit of the venous blood through the venæ vorticosæ. The anatomical relations of the venæ vorticosæ in man especially favor such a venous stasis, since they pass obliquely through the sclera and valve-like are compressed when the tension rises.

The result is a congestion in all the veins that empty into the venæ vorticosæ. This stasis is well marked in the ciliary body, the iris, and the circumcorneal veins, and is the cause of the circumcorneal injection in glaucoma. Even the veins of the conjunctiva may be congested and cause a chemosis. As a result of this venous stasis there are an

excessive exudation of liquid into the vitreous and an emigration of red and white corpuscles, causing a more or less pronounced opacity of the media. The tension is by this still further increased and the venæ vorticosæ more compressed. The glaucomatous, vicious circle is completed, and the glaucoma does not undergo retrogression (glaucoma inflammatorium evolutum).

In glaucomatous increase of tension three factors are at work: the paralysis of the muscular network of the choroid, causing a slowing of the currents of liquid; the channels of exit which, in consequence of the retarded flow, through the deposit of the formed elements, become blocked and cause a retention of the ocular liquids; and finally, the anatomical arrangement of the venæ vorticosæ, which are compressed by the increased tension and thus lead to a general venous stasis with all its results. Each of these factors may affect different persons in different ways and thus give rise to different manifestations of glaucomatous increased tension. The paralysis of the muscular network may be more or less intense. It may develop quickly or require years. The channels of exit may present great variations: sometimes they are wide and straight and are blocked with difficulty, and sometimes they are narrow and tortuous and are readily blocked. Sometimes the mouths of the veins are readily compressed, and again they remain free and open. When all these factors work together in one and the same unfavorable direction, arise the stormy acute inflammatory malignant glaucomas which resist every therapeutic effort and destroy the eye within a short time. If the conditions are less favorable, inflammatory glaucoma may of its own accord or from eserine treatment pass off altogether or pass over into simple glaucoma. When the conditions for venous stasis are relatively good, it may happen that the circulatory disturbances appear in a very mild form, or are absent as in simple glaucoma. In fine, all the manifold forms in which acute and chronic glaucoma appears may be explained readily by the various ways in which the factors work together or in opposition.

In regard to the symptoms of inflammatory glaucoma, I

have supposed the disturbance in circulation to be a result of the venous stasis. This stasis may in certain acute cases extend to the conjunctiva and give rise to chemosis. An injection more or less pronounced of the circumcorneal veins is always present. The venous stasis in the iris and ciliary body causes an increased transudation and a haziness of the media, but excites no inflammation. One of the erroneous ideas in regard to glaucoma that has prevailed since Graefe's day is that the disturbances of circulation in glaucoma depend upon an inflammation. In fact, however, none of the symptoms which characterize inflammatory iridocyclitis is found in acute glaucoma, excepting only the circumcorneal injection, which here is of a different character. The pupil is not small, synechiæ are absent, the cloudiness of the aqueous differs from that in iritis, and the "inflammatory" symptoms vanish in a few hours under the use of eserine, which certainly would not be the case if an inflammation existed. The name inflammatory glaucoma is therefore misleading, and for it should be substituted congestive glaucoma.

In congestive glaucoma, the diminished sensibility of the cornea, the shallow anterior chamber, and the immobile dilated pupil are the results of the increased tension of the vitreous and the paralyzing effect of the pressure upon the ciliary nerves.

The diminution of vision in congestive glaucoma is also to be regarded as a result of the increased tension. The cloudiness of the media has its effect on the diminution of vision, but it is not the sole factor, for often the vision is very poor when the cloudiness is not excessive, and after the use of eserine or the performance of iridectomy the acuteness and the field of vision greatly improve, indicating that the increased tension had affected the rods and cones.

In acute congestive glaucoma, the vision always improves as soon as the tension is reduced to normal, provided that from the long continuation of the increased tension the rods and cones have not suffered materially. The disturbance of function in acute congestive glaucoma, at least at first, is always of a functional nature.

In simple glaucoma, the matter is somewhat different: here the disturbance of function, when it has gone so far that central vision and the field of vision have been reduced, always depends upon an atrophy of the corresponding nerve elements. This difference in the two forms of glaucoma depends upon the difference in development in congestive and in simple glaucoma.

In simple glaucoma, the atrophy is chiefly in the inner layers of the choroid—in the chorio-capillaris. The nervous elements—the rods and cones—are early affected and with the insufficient nutrition soon are destroyed. If with this is associated an atrophy of the elastic fibres of the retina with excavation of the disc, the nerve fibres also begin to be affected, and this hastens the diminution of vision. In this way the process, without other disturbances than a continuous decrease in the functions of the eye and an increasing excavation of the disc, or, in other words, under the form of simple glaucoma, may for a shorter or longer period, often as long as several years, continue until it has caused an atrophy of the outer layers of the choroid, or extended to the layer of larger vessels and caused a paralysis of the muscular network of the choroid, by which the circulation is checked and tension rises. The acute glaucoma has thus changed its nature and passed over into the congestive form.

If the atrophic process begins in the outer layers of the choroid without involving the chorio-capillaris, there arises an acute congestive glaucoma which, if relieved, can recede to the normal function of the eye and an apparent restitutio ad integrum. Since the chorio-capillaris is healthy, the retina functionates normally and the acuteness of vision remains normal. The eye may therefore, despite a congestive glaucoma, if a successful iridectomy is done, present for years the appearance of a healthy eye. The atrophic process continues, however, rapidly or slowly, and finally affects the chorio-capillaris and with it the nervous elements of the retina. The disease then assumes the character of a simple glaucoma.

Whatever the nature of this atrophic process may be cannot be said offhand, since the pathological examinations

available give no information in this regard, mostly having been made in old cases, and the chief attention given to the ligamentum pectinatum, the other parts of the eye receiving little attention. Yet the atrophy of the choroid is one of the most constant findings. This atrophy affects different layers of the choroid in different persons and even different areas. In whatever form it appears, it has usually a very slow course. Yet the course may be rapid, as in the following case.

The wife of a laborer, fifty-seven years old, presented herself some years ago at our clinic with an acute congestive glaucoma of the right eye of two days' duration and causing great pain. The symptoms were not very marked. The circumcorneal injection was slight, the cloudiness of the media slight, the tension greatly increased, and V = fingers at 4*m*. The anterior chamber had its normal depth, the pupil was dilated, and the iris in its lower part was greatly atrophic. An iridectomy upward was made at once with good result. The left eye, carefully examined, seemed normal in every respect, and the iris responded promptly. A few days after the operation on the right eye, there was an acute attack of glaucoma in the left. This came on at 11 o'clock at night, and when I saw the patient at noon on the following day the iris was quite atrophic, so that the lens everywhere shimmered through. Here a complete atrophy of the iris had taken place in less than twenty-four hours. It was impossible for an inflammatory process to have brought about this atrophy. The only possible explanation is that a trophic nervous influence existed, and this was probably also the cause of the atrophy of the choroid. Where the nervous cause is to be sought, whether in the sympathetic, as Abadie believes, or elsewhere, I am not prepared to say.

From what has been said, it is evident that the prognosis of glaucoma in general must be considered bad, because we have no remedies which will act upon this atrophic process. But since the process differs greatly in different cases, so the prognosis differs greatly. In simple glaucoma, the prognosis is always bad. In congestive glaucoma, iridectomy or the use of eserine may relieve the tension in many cases and restore the normal circulatory conditions in the eye. The

atrophic process in the choroid continues, however, extending to the chorio-capillaris, and blindness is here also the final outcome. But this may be delayed for many years.

We are unable to say whether the atrophic process in the choroid may cease spontaneously, but it is improbable.

In regard to the treatment of glaucoma, we have not yet been successful in finding a sure means of checking the atrophic process in the choroid. The question whether Jonnesco's sympathectomy has a future cannot yet be answered, since the observations are few and the reports contradictory. Personally I have had no experience with it. But, on the contrary, I believe that I have found in repeated injections of strychnine a measure to prevent the spread of the atrophic process. In simple glaucoma, with little or no increase in tension, strychnine injections, one a day for ten to twenty days, repeated three or four times a year, have enabled me to preserve the vision for many years, although in more than half the cases the diminution of vision has continued unchecked.

In simple glaucoma, there is no indication for iridectomy as a curative procedure; but since an acute congestive attack may come on in a case of simple glaucoma, iridectomy is of prophylactic value, and the operation is more easily done in the quiescent stage than in an acute attack.

Iridectomy, as in v. Graefe's time, remains the sovereign means of reducing increased tension. Its action, however, is not understood, and has been interpreted by different writers to suit their theories of glaucoma. The adherents of the retention theory attribute its action to the opening of the filtration angle and the facilitation of the outflow of liquids from the eye. Since this theory has obtained the most general acceptance, we may discuss it here. In order to be efficacious, the iridectomy must be as peripheric as possible. The lance knife must be entered 1*mm* outward from the sclero-corneal junction and passed into the anterior chamber parallel to the surface of the iris, so that the section will lie close to the root of the iris. When the root of the iris is adherent to the cornea, it is absolutely impossible to enter the anterior chamber in this manner. The section

must lie farther forward in the cornea. When the iris is drawn out with the forceps, only the free portion comes, the root of the iris remaining behind. This is seen in eyes examined microscopically. In such cases there can be no hope of releasing the root of the iris and restoring the filtration angle. Such adhesions, however, are found only in old cases of chronic congestive glaucoma in which there can be no hope of curing the glaucoma and restoring normal conditions, and I dare to assert that no one has cured such a case of glaucoma. In all the cases of glaucoma in which iridectomy has been of service, the angle of the interior chamber must have been open, otherwise the lance could not have entered.

The retention, therefore, does not depend upon the fact of the angle being more or less open, so long as it is sufficiently open to permit the passage of the knife; for if this is possible liquids also may pass. The check to circulation is thus not in the angle itself, but in the surrounding tissues and fine canals which lead through these tissues to the circumcorneal veins. When a portion of the iris is removed, it does not follow that the channels of exit are opened and resorption increased, but rather the contrary, since a portion of the absorbing surface of the iris has been lost. This is shown by the simple clinical observation that blood in the anterior chamber is readily absorbed where the iris tissue is preserved; but in the coloboma, particularly when this is downward, the blood may remain unabsorbed for weeks. This certainly does not indicate that the iridectomy has opened the filtration angle.

After a successful iridectomy in acute congestive glaucoma, it is always the so-called inflammatory symptoms—that is, the disturbances of circulation—that first pass off, and the beneficial effect of iridectomy lies in its effect upon the circulation. The most probable explanation of the action of iridectomy in glaucoma is that given by Exner. In the operation a portion of the iris is removed up to its ciliary attachment, and the short arterial twigs which proceed from the major arterial circle of the iris have the opportunity to form direct anastomoses with the corresponding venous

twigs. The arterial current can thus pass directly into the veins without being obliged to pass through the fine-calibred and rich capillary network. The resistance in the blood current is therefore diminished, and the tension sinks not only in the short arterial stumps, but also in the entire major circle and in the choroid. Iridectomy thus works, as Knapp expresses it, as a safety-valve.

The increased intraocular tension is, as before stated, a result of the slowing of the currents in the eye, which occurs when the contractility of the choroid diminishes in consequence of the action of the atrophic process upon the muscular network in the layer of larger vessels. The formed elements in the ocular liquids then become deposited in the fine channels of exit and block them and the tension rises. If now the anatomical relations of the venæ vorticosæ are unfavorable, it readily happens that compression results with venous stasis, which again increases the tension and completes the vicious circle which causes congestive glaucoma. The purpose of the iridectomy, therefore, is to break this last link in the chain and prevent the venous stasis.

If the iridectomy is successful in preventing the development of the venous stasis, the tension becomes normal or but slightly increased, according as the channels of exit have become patulous or not.

The glaucomatous process is not at an end after the iridectomy, but, as a rule, progresses. As a rule, many of the patients operated on for acute glaucoma complain of slight temporary attacks of cloudiness of vision and increased tension without permanent injury to the vision.

In this way the condition may continue for years, until a fresh acute attack occurs, or the disease gradually assumes the character of a chronic congestive glaucoma with continued diminution of vision; or there may be no attacks, the eye appears normal, and its functions continue normal for years. But eventually central vision becomes poorer, the field contracts, and the nerve head becomes excavated.

When a fresh acute attack occurs, or the disease assumes the character of a chronic congestive glaucoma, an iridectomy opposite the first may again give relief and the eye

functionate again. Finally, however, vision decreases more and more and the tension increases, with severe pain. Posterior sclerotomy, perhaps repeated, may give temporary relief, but at last we can only relieve the patient by enucleation or evisceration.

An anterior sclerotomy may doubtless in many cases relieve a glaucoma, but in most cases its action is nil. When the wound heals, the effect of the operation is at an end, and an iridectomy has usually to be made.

In cases in which anterior sclerotomy is of no benefit, posterior sclerotomy may be tried. With a cataract knife, a longitudinal section 3 to 4*mm* long is made through the sclera in the equatorial region. In order to be sure that the wound will not close immediately, a second section may be made perpendicular to the first. Thus there is formed a triangular wound which remains open several days and allows the vitreous fluid to trickle through. The eye remains soft, and there is time for the circulatory disturbances to pass off. These operations, particularly the posterior sclerotomy, are indicated rather in the old cases that have run their course, in which there is no question of the restoration of vision, but a desire to relieve the patient's pain.

In fresh cases of acute congestive glaucoma in which for any reason iridectomy cannot be done, frequent instillations of eserine act best in reducing the tension. Sometimes a single or several instillations are sufficient, but usually many are necessary. I have sometimes had to instil eserine forty to fifty times in the course of twenty-four hours in order to reduce the tension. As a rule, I begin the treatment of a glaucoma with frequent instillations of eserine, and after becoming convinced of its futility I proceed to operation. In this way the congestive symptoms are checked and the operation can be more satisfactorily performed and a better result obtained than when one operated in an acute glaucomatous state.

Eserine causes a contraction of the muscles of the eye. Through contraction of the tensor of the choroid and probably also through its action upon the muscular network about the choroid, this membrane is rendered tense and the

tension falls. The pressure upon the sclera is relaxed and the mouths of the venæ vorticosæ open. The venous blood escapes from the eye and the circulatory disturbances are relieved. When the atrophic process in the layer of large vessels has gone so far that the muscular network and the tensor are completely paralyzed, eserine has no effect. Unfortunately, as I have remarked, this is often the case. The contraction of the pupil has no curative effect, but may furnish us an indication that the contractility is preserved and that we may hope for a favorable effect upon the deeper-lying muscular fibres.

In simple glaucoma in which the atrophic process is confined exclusively to the chorio-capillaris, eserine has no effect, but as it is not possible to say whether the process is confined to one layer alone, we may take it for granted that different layers are affected, though in different degree, and hence in most cases of simple glaucoma a continued use of eserine acts well and in many cases prevents increase of tension.

A CLINICAL CONTRIBUTION TO TRAUMATIC ENOPHTHALMUS.

By Dr. PURTSCHER, Klagenfurt.

Abridged Translation from Vol. XXXVIII., 2 (1898), of the German Edition, by Dr. Ward A. Holden.

ALTHOUGH in recent years a fairly large number of reports of traumatic enophthalmus have been published, the views in regard to its true nature are still divergent, as the latest communications show.

The chief reason for this difference is doubtless due to the fact that the cases reported are in reality of different nature, which is true of other clinical pictures, as particularly Schapringer and Th. Beer have pointed out.

We should exclude here the cases in which severe trauma has destroyed a portion of the skull about the orbit. Such cases I have repeatedly observed. In one case a dog was said to have struck a miner in the superior maxillary region, so hard that the upper and anterior portions of one superior maxilla were completely shattered, so that the ball sunk into the antrum of Highmore. In a second case, a kick from a mule's hoof produced a similar effect. It does not surprise us that under such circumstances a direct extensive dislocation of the ball takes place. In a strict sense even those cases should be excluded in which, according to Gessner's view, a cicatricial shrinking of the orbital tissues following inflammatory processes comes on after a trauma in the region of the eye. Still these cases differ little from the pure cases in which, while the walls of the orbit remain normal, a non-inflammatory sinking of the eyeball appears

more or less quickly, which Beer refers not to paralysis of the sympathetic or pressure atrophy, but simply to trophic disturbances in consequence of the injury of peripheral nerves.

Every new case-history may assist in clearing up the question, and a case which came under my observation follows:

Anton Z., aged forty-six, was admitted to the hospital Oct. 14, 1897. He had previously enjoyed good health until five weeks before, when being struck by a cow's horn he suffered an injury in the region of the superior wall of the orbit, and a second in the region of the inferior wall. These wounds were sutured by the local physician. He was dazed for a time but did not lose consciousness. There was some hemorrhage from the nose. The eye was so swollen that the patient could not see out of it for a week.

The patient came with the complaint that he could not raise the upper lid properly and that he still had pain in the depth of the orbit.

Status præsens.—At first glance one noticed the smallness of the left palpebral aperture, and the deep location of the ball in the orbit, as if the patient were wearing an artificial eye. Noticeable also is the circumbulbar sinking in of the lids, and the left cheek seems to be less developed than the other. When more closely examined, one sees a scar, partly keloid-like, beginning near the incisura supraorbitalis, or the superior margin of the orbit, and extending nearly to the outer margin of the orbit. A second, shorter scar, similar in character, is seen at the inferior margin of the orbit. Neither scar is adherent and both move readily over the bone.

The length of the palpebral aperture is *22mm* and its height *5mm* as compared with 25 and *10mm* on the right side. No deformity of the bony margin of the orbit can be felt, but the patient complains of pain when firm pressure is made against the superior margin. There is furthermore a marked anæsthesia in the region of the supraorbital nerve, but the eye itself is not anæsthetic. The ptosis leaves only the lowest third of the pupil clear, and when the other eye is wide open there is but slight elevation of the left upper lid. The eyeball is sunken at least *3mm* into the orbit. Conjunctiva, lachrymal sac, and the external portions of

the eye are entirely normal. The cornea in its primary position is 1½*mm* higher than the right. The pupils are of equal size and each reacts normally.

R, V = $\frac{3}{10}$; with 1.5 = $\frac{3}{6}$.

L, V = $\frac{3}{10}$; with 1.5 = $\frac{3}{6}$.

Accommodation intact.

Movement of the eyeball inward of normal extent, movement downward practically abolished, yet there is a rotary movement due to the action of the trochlearis. The excursion outward is of moderate extent and with it the upper end of the vertical meridian inclines outward. Movement upward very limited.

The patient does not complain of diplopia, yet after raising the lid and holding a red glass before one eye diplopia occurs, but the significance of it is uncertain on account of the complicated paralyses.

The color fields are normal, and the fundus is normal in each eye.

The experiment of subcutaneously injecting pilocarpine for diagnostic purposes with reference to the sympathetic, suggested by my colleague, Dr. Pichler, resulted negatively.

After ten days the patient was discharged as there was no hope of improving his condition.

To this case I can add a brief sketch of another in respect to which I find myself in the same position as Beer with his second case.

The patient could be but briefly examined because of lack of time and did not keep his promise to return.

A man of about twenty while serving in the cavalry was kicked in the right frontal and temporal region a year before. There was no swelling in the region of the eye, yet he became suddenly and permanently blind and has since been able to open the eye only with difficulty. He stated that the eye at once became smaller. At the time of the examination the patient complained of diminution of vision in the left eye.

Externally no scar. Right eye quite closed, but with an effort it can be opened fairly wide. The eyeball of normal appearance is so deeply sunken in the orbit that the patient has worn an artificial eye over it. Movements in all directions normal; no deviations. V = 0; complete atrophy of the right optic nerve. L ophthalmoscopically normal. V = $\frac{3}{6}$.

Before proceeding to the discussion of these cases, I wish to refer to some of the more recent reports which have appeared since the fundamental paper by Th. Beer from Fuchs's clinic.

The first of the more recent cases is that of Georg Cohn[1]:

A man was struck on the eye, and suffered a fracture of the superior maxilla, by a tame deer which had suddenly become wild.

The latter factor rather rules the case outside of our category, although the author believes that besides the fracture a cicatricial contraction of the orbital connective tissue contributed to the production of the enophthalmus.

Two recent cases reported by Schapringer[2] follow.

Case 1.—A coachman, aged seventeen, was kicked in the eye four months before.

Ptosis. Enophthalmus. A non-adherent scar at the lower margin of the orbit. Cheek somewhat depressed. Sensibility undisturbed. No vertical deviation; good mobility. Pupil of medium size. Partial atrophy of the optic nerve.

The author assumed an extensive fissuring of the walls of the orbit.

Case 2.—A woman of thirty. At the age of eight she fell from a wagon.

Ptosis. Enophthalmus. Normal mobility. No scars about the eye. Pupil normal. Vision good.

The author calls attention again to the importance of injury of the peripheral nerves as the probable cause of traumatic enophthalmus, and recognizes the significance of such injuries also for the trophoneuroses in the sense of Beer.

There should also be mentioned a case of Schwarzschild's[3] referred to by Schapringer:

A coachman of twenty-two had fallen from a vehicle at the age of eight. Wound of the left eyebrow. The condition was regarded as the immediate effect of the trauma suffered, yet definite statements as to the exact time are wanting.

No mention was made of ptosis, nor yet of its absence. Vision and interior normal. Undiminished mobility, yet slight atrophy of the affected half of the face.

This case is not a typical one, since the enophthalmus of *6mm* became an exophthalmus of *6mm* whenever the head was bent forward, as in similar cases described by Robert Sattler, Gessner, Sergent, and others.

A further case, like Cohn's from being struck with a deer's horn, was demonstrated by Fuchs.[4]

The patient, a forester, had a ptosis of *4mm*. The eye was *2mm* lower than the other and was somewhat sunken into the orbit.

There was anæsthesia of the left cheek, under lid, nose, and lip; and diplopia due to paralysis of the inferior oblique. This diplopia was successfully relieved by tenotomy of the right superior rectus.

The cause of the enophthalmus was thought to be a fracture of the roof of the orbit.

Strictly speaking, perhaps this case does not belong in our category.

Another case was observed by Oliver[5] (cited by Neulen):

A weaver, forty-seven years old, had been struck by a shuttle in the left eye five weeks before. The eye and the surrounding part were then inflamed. The patient complained of diminution of vision in this eye. Enophthalmus of 4–5*mm*. Palpebral aperture *3mm* narrower than the other. Almost complete paralysis of the superior rectus; paresis of the inferior oblique. Fracture of the bone not to be made out.

The case reported by Denig[6] is of importance:

A mason, aged twenty-six, fell from a height of *1m* and struck on the left side of the head. No disturbance of consciousness, but vomiting, and hemorrhage from the right ear. Diplopia soon came on.

Ten days later the patient noticed that his left eye had become smaller.

Enophthalmus of *3mm* with slight ptosis. Bilateral paralysis of the abducens. Left pupil considerably dilated but responsive. No scars and no depression. Suppurative otitis media. Paræsthesia of the left eye, cheek, and left half of the nose. Unilateral perspiration. Left cheek flattened. Otherwise normal. The enophthalmus, flattening of the cheek, and paralysis of the abducens continued.

Denig believed that the enophthalmus as well as the flattening of the cheek depended upon the fifth nerve proper and not upon its sympathetic fibres. The paralysis of the sixth nerves he attributed to a fracture of the base.

Denig's case, as he states, was the first until then observed in which enophthalmus followed injury of the skull. In the other cases there were injuries of the face.

Another case is that reported by Neulen [7] from Schirmer's clinic:

A patient, aged sixteen, was kicked eight weeks before and remained unconscious for twenty-four hours; after this there was amnesia as to what had occurred before. The left eye and left upper jaw were struck. On the cheek was a wound in the skin, which was sewed up.

In the first week there was much swelling. After this had passed off the patient noticed that he could only recognize movements of the hand with the left eye.

There was suppuration in the wound and several incisions were required.

Two large scars were found, one at the lower margin of the orbit in connection with a fistula which led to rough bone; the entire lower margin of the orbit was prominent and thickened. Ptosis of *6mm*, enophthalmus of *3mm*. Movements of the eyeball outward somewhat limited. Pupil slightly contracted.

Nothing is said of the acuteness of vision, but it must have been poor since there were two ruptures of the choroid and a white almond-formed patch at the macula.

Neulen in his dissertation concludes with Lang that traumatic enophthalmus is due to fracture of the orbital wall.

Two cases reported by Maklakoff, Jr.,[8,9] follow:

Case 1.—A man of sixty-seven received a blow eight months before which caused a splitting of the lower lid. The swelling lasted two weeks. Then ptosis and sinking-in of the eyeball were observed.

No fracture could be made out and no irregularity of the face. No increased sensibility. No anæsthesia. Above and below a sort of symblepharon: dilated pupil, luxation of the lens, and opacities of the vitreous. V with + 12. D = 0.7.

There were muscular pareses.

The author, supporting his position by an observation of Jaboulay, who observed enophthalmus after resection of the sympathetic, believed that the lesion was in the region of the ciliary ganglion.

His second case was reported a year later:

CASE 2.—A cook, aged forty-two, received a blow from the fist of a thief on the right cheek, causing a wound in the skin. There was swelling at first and afterwards diplopia.

He found a long scar along the margin of the orbit. Sensibility diminished on the lids, cheek, forehead, and cornea. Pupils equal; accommodation normal. Paralysis of the external and inferior recti and of the inferior oblique.

The author locates the lesion at a point in the orbit about 4*mm* anterior to the optic foramen, close behind the ciliary ganglion. The primary cause he believed to be a hemorrhage from the ophthalmic artery. Above it lie the fibres of the sympathetic, outward the abducens, below the naso-ciliaris, farther below the inferior branch of the oculomotor, and farther above the superior branch. Hence one can understand why the internus remains most protected while the other nerves are so frequently affected. He distinguishes three groups according to the severity of the lesion.

A case was reported by Darier[10] simultaneously with his second case, which differed somewhat from it but was of particular interest from a therapeutic point of view:

A young man had a severe fall from a horse, and lay comatose for two weeks. Injuries of the face and probable fracture of the base. Hemiplegia and facial paralysis of the same side. At the beginning there was ptosis, and later an abnormal elevation of the lid. The eye moderately turned out. The pupil at first dilated, later normal. Only the abducens preserved its normal function. Accommodation preserved. $V = \frac{1}{2}$.

Darier assumes as the cause of the enophthalmus that from the severe contusion of the entire contents of the orbit an inflammatory condition of irritation of the retrobulbar tissue was set up, ending in the formation of a scar

which drew the eyeball backward and hindered the action of the muscles.

He agrees with the views of Gessner.

Darier deserves the credit of first relieving the enophthalmus by making tenotomies. A tenotomy of the tendon of the external rectus improved the condition, and then, in order to increase the effect, he tenotomized the other recti muscles with satisfactory effect, both from a cosmetic and from a functional point of view.

In the same number of the *Clinique ophtalmologique* there are two other references to cases of traumatic enophthalmus.

One by W. E. Brunner,[11] whose patient was a man of thirty-three, who fell, breaking the nose and the zygoma. Ptosis. The eye could not be moved upward. Pupils equal.

The author considered the ptosis as the mechanical result of the deep location of the ball.

A second by Roberts.[12] Fracture of the roof of the orbit from a horse's kick. Ptosis. Abducens paralysis. Death from meningitis.

Neither of the cases seems quite pure.

Strictly considered, of the new cases only those reported by Schapringer, Denig, Oliver, Neulen, and Maklakoff are quite free from criticism, perhaps also Darier's case, although it was complicated. The cases of Cohn and Roberts are to be absolutely excluded.

All the cases of traumatic enophthalmus not due to gross fractures of the orbit have in common: 1, the traumatic origin; 2, the enophthalmus; and 3, more or less marked ptosis.

It is evident that after the action of severe force upon the region of the eye or the skull other symptoms may be found. Thus there is rarely lacking a cut or a scar on the skin in the region of the orbital margin. An accompanying symptom almost as frequent is paralysis of ocular muscles, which, as Beer said, need not be orbital. In three cases, furthermore, there was dilatation of the pupil, and in one contraction. The accommodation seemed, as a rule, to be intact.

A remarkable accompanying symptom is an anæsthesia of

some branches of the fifth nerve, and another remarkable symptom is the unilateral atrophy of the face. Unilateral sweating, also, has been observed. Besides these, we find partial or total atrophy of the optic nerve, facial paralysis, and hemiplegia. In one case the enophthalmus became an exophthalmus when the head was bent forward. Finally in one case there were ruptures of the choroid, and in another opacities of the vitreous and luxation of the lens.

Maklakoff calls attention to the frequency of paralysis of the external rectus in these cases, and from this concludes that hemorrhage from the ophthalmic artery is capable on the whole of explaining the symptoms in traumatic enophthalmus. The abducens, because of its nearness to the artery, is particularly often affected.

On the contrary, I would state that, in the literature of injuries of the head, paralysis of the sixth nerve is much more frequently noted than paralysis of any of the other ocular nerves, even when the lesion is nuclear or basilar, so that its frequency in traumatic enophthalmus cannot be regarded as proving the orbital location of the lesion. This was made out in Denig's case only.

Yet on the whole we can say that the typical clinical picture of pure traumatic enophthalmus perhaps is not always due to the same cause. For there is not in all cases a fracture of the orbital wall enlarging the orbital cavity, nor is there in all cases a cicatricial contraction following orbital inflammation. There are, however, apart from fracture, some direct mechanical factors which are capable of causing a slight degree of dislocation of the ball,—thus, for example, a direct or indirect solution of continuity or loosening of the fascia of the orbit, or a loss of tension in the girdle of the obliqui. I believe that some weight must be placed in this idea. Tenotomy of the recti muscles causes exophthalmus, and Darier obtained by tenotomies a satisfactory correction of the enophthalmus in his case.

The possible rôle of the sympathetic was first suggested by Schapringer. No one will deny that lesions of the sympathetic will cause ptosis, but it must not be forgotten that the most important factor in producing the ptosis in these

cases is the sunken location of the eyeball. Just as a protrusion of the ball after tenotomies will raise the lid, so an enophthalmus will allow it to droop. It is not surprising, then, that ptosis is found constantly in cases of enophthalmus; only Darier's case is an exception, for in this case the ptosis later gave place to an elevation of the upper lid. The ptosis may be considerable, because the levator may be paralyzed as well as the other muscles.

The explanation of the ptosis does not hold good for the defects in mobility of the ocular muscles. Beer called attention to the fact that through the enophthalmus itself a certain limitation of mobility of the recti might be brought about by the lessened distance between their origins and insertions with the consequent relaxation. But all the defects cannot be laid to the enophthalmus, since clinical experience shows that in most cases individual muscles alone are affected, indicating a direct lesion of the muscle or its nerve. Naturally a direct lesion must be held responsible for anæsthesia of the branches of the fifth and for facial paralysis.

From what has been said it appears that there are cases whose characteristics depend solely upon the traumatic enophthalmus. To interpret these pure cases we must rely upon the neurotic hypothesis of Beer, which clearly explains the sinking-in of the ball as being due to absorption of the orbital cellular tissue caused by lesion of trophic peripheral nerves. The atrophy of one half of the face and the anæsthesias clearly support Beer's idea, and Denig's case shows the direct involvement of the fifth nerve.

In accepting this explanation, other possibilities cannot be altogether excluded. The explanation on the ground of cicatricial retraction is not very probable, since the extensive scars are never adherent, nor is the bony wall of the orbit deformed. There is no adequate reason for supposing a particular injury of the obliques, and against paralysis of the sympathetic are the normal pupil and the similar condition of the two halves of the face after the subcutaneous injection of pilocarpine.

The most probable explanation, then, is that of trophic disturbances of the orbital tissues following injury to the nerves.

I do not feel qualified to state whether the disturbances of the ocular muscles are in the orbit or behind it. The fact that the intrinsic muscles are intact would speak rather in favor of the latter.

The second case, with optic-nerve atrophy, would indicate a fracture of the orbit, so that an exit of orbital contents could be regarded as probable. The case shows, however, that different causes may give rise to the same clinical picture or at least produce symptoms for which a uniform explanation is impossible. We lack, as Beer stated, autopsy reports.

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12. ROBERTS, J. B., Traumatic Enophthalmus from Fracture of the Frontal Bone; Trephining; Death. *Arch. f. Augenheilk.*, xxxiv., 3, p. 258.

THE SO-CALLED EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA AND OF ITS BRANCHES.

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SINCE von Graefe in 1859 described the so-called embolism of the central artery of the retina, many cases have been observed, and the addition of cases of embolism of single branches has completed our knowledge of the clinical picture presented by the disease. The symptoms are typical and always the same in their important characteristics.

After one or more transitory obscurations of one eye, rarely of both, there is a sudden attack of blindness with no external cause and accompanied by no other subjective symptoms. The blindness remains complete, or, after a short time, the vision returns, very rarely back nearly to normal, usually to only a trace in the temporal portion of the field. Sometimes both eyes are affected simultaneously, but in different degrees, so that while one regains more or less normal vision, the other is permanently impaired. Sometimes the second eye is affected years after the first has been lost. Ophthalmoscopically the media are clear; the retina at the posterior part of the fundus presents a dense, white opacity with radiating striations about the pallid papilla, uniformly diffuse in the region of the macula, in the midst of which appears a red spot with regular margins. The opacity extends more or less toward the

periphery, gradually becoming thinner, and is bordered by flame-shaped spots. The blood columns, particularly in the arteries on the papilla and in its neighborhood, are smaller than normal, so that the vessel often appears as a fine red thread, or as a gray cord from which the blood stream emerges, spreading out in a conical form. More peripherally the blood columns are broader, but yet narrower than normal. Sometimes irregular constrictions can be seen where the blood columns begin to expand. In the course of time the vessels become better filled with blood, but they seldom again attain their normal calibre. Then a uniform diminution gradually takes place, which brings about the final condition. This general picture may be modified by the presence of little spaces supplied by the cilio-retinal vessels remaining unaffected, as well as by the involvement of a single branch alone, when the opacity will be confined to the part of the retina supplied by that branch, and a corresponding defect in the field of vision will be produced.

Such a clinical picture can be produced only by an obstacle which suddenly arrests the circulation of the blood in the affected artery and then after a time allows it to recommence. Such an obstacle must be in the central artery of the retina or in one of its branches, because if it were situated in the ophthalmic artery anteriorly to the point where the central artery is given off, the collateral circulation would be sufficient to prevent any serious damage to the retina, and if the supply to a larger vascular field were cut off, the correct diagnosis would be indicated by other symptoms which could not be overlooked.

This obstacle was supposed by von Graefe to be an embolus, but soon doubts began to be expressed in regard to the embolic nature of the lesion because certain symptoms did not accord with this supposition, and various theories were advanced which found few adherents and were usually open to severe criticism. Such lesions as inflammation of the retrobulbar cellular tissue, retrobulbar neuritis, and tumors must be excluded, because they would cause other symptoms in addition to the arrest of the circulation in the central artery and the partial necrosis of the retina from the

anæmia thus produced. The theory of a hemorrhage or an exudation into the sheath of the optic nerve was disposed of by von Graefe himself. Magnus suggested that an acute amaurosis could be caused by a hemorrhage from the central artery into the optic nerve so as to arrest the conductivity of the nerve fibres by laceration, crushing, or pressure, and that later, as the blood became absorbed, or was otherwise removed, conductivity could return to the fibres which had not been seriously damaged so that a part of the retina could resume its function. But, as Leber says, both a new problem is introduced in the cause of the hemorrhage and the hemorrhage itself is assumed to act in an extraordinary manner. It is to be expected that in case of such a hemorrhage the blood would follow the sheaths of the vessels or spread out among the larger nerve bundles so as to produce local injuries which would result in the formation of definite scotomata.

This hypothesis, and to a still greater degree those yet to be mentioned, necessitates that venous stasis should be a prominent symptom, but it is never present. It should also be noticed that none of these hypotheses explain the prodromata, the periods of improvement, or the circulatory conditions, and that they are not adaptable to embolism of the branches.

The interruption of the circulation has been ascribed to spasmodic contraction of the vessels by various authors, especially by Raynaud, who in 1862 described a clinical picture of periodic attacks of pallor, cyanosis, a feeling of impending death, œdema, and paræsthesia of the extremities, particularly of the fingers and toes, which frequently resulted in gangrene. He ascribed this to a spasmodic contraction of the arteries caused by a central vasomotor neurosis, which could be seen in the retinal vessels in the form of contraction of small isolated segments. But Panas states that he was unable to detect any such alternation of hyperæmia and ischæmia of the retina in any of the patients with local asphyxia of the extremities whom he examined at Raynaud's request. Although a spasmodic contraction of the muscular coats of the arteries sufficient to close their

lumen can be brought about by artificial irritation, it is not known that such a spasm ever occurs spontaneously. The unequal, short, persistent constrictions of the arteries may be ascribed much better to diseases of their walls than to spasm, for the thickening can often be seen as an opacity while the outer contour of the wall remains normal, and, according to Professor von Frey, an artificial spasm always affects the muscular coat of a vessel uniformly for a considerable distance. Vermiform contractions of the vessels, as described by Liebreich, is an erroneous interpretation of an ophthalmoscopic picture, perhaps of that here termed granular current. The group of symptoms described by Raynaud and called by Charcot intermittent lameness (intermittirendes Hinken) is commonly considered to be the result of endarteritis obliterans. Quinine amaurosis cannot be quoted in evidence, because the manner in which the amaurosis is produced has not yet been determined.

There remain only three possible forms of obstruction to the circulation which can produce the typical cases of so-called embolism of the central artery of the retina, embolism, thrombosis, and endarteritis proliferans. Before going farther, a symptom should be discussed which, in my opinion, has not yet been rated at its full value,—the granular current, or the breaking up of the blood in the blood-vessels. It was first noticed by Jaeger in 1854, and was described by von Graefe, Donders, and Cuignet. Donders saw it in conjunctival veins, Friedenwald in newly formed corneal vessels. It can always be obtained in the conjunctival and episcleral vessels when the blood is sufficiently retarded by suitable pressure. Jaeger in 1876 gave an accurate description of this phenomenon which I will summarize. At first the movement of the contents of the vessels becomes observable, presenting an appearance as if fine sand was being driven through a glass tube with great swiftness. The more the rapidity of the current is reduced, the clearer the granular appearance becomes, until there is a separation of the blood into red and white cylinders which, wherever the vessel branches, accommodate themselves to the corresponding diameters, though many times the red cylinders pass by

a little twig, particularly when it forms quite an obtuse angle with the main trunk, and this is left filled with a colorless fluid. When a red cylinder passes from a small vein into a larger one, it adapts itself to the increased diameter, becoming shorter and broader. Sometimes all movements cease and the cylinders remain motionless. When circulation is restored, there is often a pulsating fluctuation, the cylinders moving by starts. As the circulation increases in rapidity, the current passes from coarse-grained to fine-grained, until finally the vessels again hold uniform red columns and show normal reflexes from their walls.

Almost all authors who have studied this phenomenon agree that retardation of the circulation is sufficient to account for its occurrence. While the blood is in rapid motion the cellular elements form a pretty even mixture in the plasma, but when the circulation is considerably retarded the red blood corpuscles gather together in larger or smaller masses between which the plasma appears as a bright yellow fluid. As long as the current is fairly swift, the masses formed are small, so that they create an impression like that made by flowing sand, particularly in the larger vessels. As the current becomes slower, larger masses are gathered together into dark red cylinders separated from the light yellow plasma by sharply indented boundary lines, which resemble the fracture lines of cast-iron bars. The result of the agglutination of the red blood corpuscles is that when the current is slow they slip by a small twig given off from an artery at nearly a right angle, and as they are not tightly joined together they spread out and form a shorter mass when they pass from a small vein into a larger one.

Some similar condition must be present when the circulation is completely stopped and also after death. Knapp made the inexact statement that at death the arteries are emptied and all the blood segregated, some in the capillaries, but mostly in the veins. Von Schulten states more accurately that at the moment of death the vessels become emptied of a great part of their contents, which assume a venous character. But it is peculiar that although a tension of 8 or 10*mm* prevails in the eye, much blood often remains

in the retinal and choroidal vessels whatever the cause of death may have been. Usually the columns of blood in the vessels are broken in several places. Schnabel and Sachs say that the papilla grows pale and its vessels attenuated shortly before death, and that immediately after death its vessels become light threads, often with red lines in the middle, while outside the papilla they are only partially filled.

The vessels can be said truly to be empty only when their physiological contents have been removed, and that is possible practically only with collapse of the walls. I have referred in a previous paper to the importance of this fact with regard to microscopical examinations, but point it out again because until very lately the expression, "the vessels are quite, or in places, empty," has been often met with in descriptions of ophthalmoscopic examinations, while the appearance referred to may be the result of collapse, of contraction, of pathological thickening of the walls of the vessels, or of the filling of the lumen with plasma, conditions which are very different and deserve to be differentiated.

Usher found a granular current at the moment of death, the blood columns broken to pieces afterward, and I have myself seen these post-mortem appearances in the eyes of rabbits and cattle. Nettleship, in a microscopical examination of an enucleated eye, found the blood column broken in many places, a phenomenon which he considered a post-mortem change. This filling of the vessels in places with agglutinated red blood corpuscles and plasma is naturally found in microscopic sections in vessels cut longitudinally.

In experimental section of the optic nerve and the central and posterior ciliary vessels, Wagenmann always found the blood columns broken to pieces as soon as the circulation was interrupted. Later, when the circulation was restored, there was a granular current which disappeared as soon as the current was swift enough to prevent agglutination. He also cited clinical cases in which the same picture was present after traumatic section of the central artery of the retina. The same appearance can be produced in the living eye by pressure properly applied so as to diminish the quantity of

blood that enters and thereby the rapidity of the current. Then the phenomenon is seen best in the veins against the bright background of the papilla. First comes the appearance as of rapidly flowing sand, then, with increased pressure, slowly flowing pieces and single cylinders. Then sometimes the proximal ends of the veins look like broad white bands into which the agglutinated red blood corpuscles disappear. The only explanation of this is that at this place the lumen of the vein has been reduced by the pressure to a very small slit, through which the blood flows very swiftly in a very thin and ophthalmoscopically invisible layer. With still greater pressure, the granular current may be produced in the centrifugal current of the arteries, which, under yet greater pressure, may turn and flow centripetally, when the blood may be pressed out of the entire retinal vascular system. Meyerhofer observed a similar condition in his case of embolism.

It is easily understood that agglutination takes place first in the veins, because the current is slower in them than in the relatively smaller arteries, through each of which the same quantity of blood flows in the same time. On the other hand, cases in which the granular current occurs in a single artery while the corresponding vein shows an uninterrupted blood column, can be explained by the consideration that the territories of the larger arterial and venous branches do not exactly correspond, that the vein in question contains not only the slowly flowing blood from the artery with the granular current, but also rapidly flowing blood from other arterial branches, and that the movement of the mixture is sufficiently swift to prevent agglutination. The ease with which a granular current may be obtained by pressure on the eyeball differs in different persons. It is greatest in very anæmic persons, perhaps because the diminution in the number of red blood corpuscles allows the phenomenon to become visible more quickly, perhaps because in them the red blood corpuscles possess greater power of agglutination.

The conclusion I draw from what has preceded is that circulation is present as long as the thread of blood in its course toward the periphery is not broken to pieces, or the red

blood corpuscles agglutinated, no matter how thin it may be, or how covered in places by thickening or opacity of the vessel walls. It may be possible to form an estimate of its swiftness from observation of the granular current in other cases. A complete interruption of the blood stream can be assumed only when the blood column is broken and the separated portions stand still. The absence of pulsation, either spontaneously or on pressure, is no sign that circulation is not present.

If we accept the view of Donders, that pulsation in the veins is due to the fluctuating tension caused by the pulsating entrance of arterial blood into the eye, the impossibility of obtaining it by pressure is only a sign that the quantity of blood which enters the eye with each systole of the heart is so small that it cannot produce a visible compression of the ends of the veins when they are normal. On the other hand, the theory of Donders, or of Coccius, as opposed to that of Helfreich, is supported by the fact that in many cases of so-called embolism, in which there was a marked narrowing of the lumen of the artery, a spontaneous or artificial venous pulse was at first absent, but later was re-established when the diminution of the obstacle permitted the passage of more blood, for the blood must escape normally from the veins if they appear to be normally filled.

Pulsation on pressure in arteries of normal calibre shows that there is no marked obstacle in their lumen, for if the calibre is constricted at a certain point the pulsation will appear on its proximal side, unless the walls are too rigid, but the diminished quantity of blood driven by the obstruction will be unable to distend visibly the walls on the distal side. If, however, the lumen at the constricted place should become enlarged in the course of time, a sufficient quantity of blood might be able to pass through to produce a visible distal pulsation with each systole. This same theory may also account for the pulsating movements of the arteries which can frequently be seen in healthy persons when sharp bends are present in the arteries normally and physiologically. The application of these theoretical considerations cannot be exemplified from literature, and the arterial pulse from

pressure is influenced by many other factors than the presence of an obstacle to the circulation.

It is important to take into consideration the behavior of the vessels, especially of the arteries, in order to correctly interpret the clinical picture of so-called embolism. Without exception, there is a diminution in the size of the arteries, particularly marked on the papilla and in its neighborhood, while the peripheral parts are larger. This general diminution in size of the vessels is due in part to the lessened supply of blood, but as regards other factors there are two views. Fischer thinks that after the circulation has been interrupted the blood pressure falls below the intraocular pressure, and so the blood is pressed out, while Elschnig thinks that the elastic contraction of the arterial walls expels the blood as soon as its strength exceeds that of the blood pressure. I believe both factors take part.

The fact that the larger arteries are often so remarkably small at their beginnings, Elschnig ascribes to a contraction of their walls caused by the ischæmia, and he takes the ground that the strength of the musculature of the larger arteries suffices to empty them, while in the smaller it is insufficient to do so. Without regard to the fact that ischæmia is commonly supposed to dilate arteries, I think that the musculature of the vessels is proportioned to their size, and that the weaker muscle produces the same effect on the small vessel that the stronger does on the large. Electric irritation causes the vessels to contract evenly throughout their ramifications, at least in rabbits. It is much more likely that the great diminution in size of the proximal parts of the arterial blood columns depends on arterio-sclerotic changes in the walls, an evidence of which is the fact that the wall can often be seen as a gray cord of the proper size of the artery, with a small thread of blood in its middle, while in contraction of the artery the entire cord would appear reduced in size. A normal external contour of the vessel can often be made out when the calibre appears to be unevenly constricted.

Raehlmann says that the healthy wall of a retinal vessel filled with blood cannot be seen and differentiated from the

surrounding tissue as long as the retina is healthy. This rule is not without exception, for in many cases normal vessels in a healthy retina reflect so much light that with a plane mirror and weak illumination a faint gray glimmer along one margin of a vessel can be seen, which must be a reflex from its wall. I have sometimes seen under the conditions mentioned by Raehlmann a fine gray stripe, usually on both sides of the larger vessels, which I believe to be not a reflex but the vessel wall itself, made visible on the sides by the formation of a thicker layer than appears elsewhere. Schneller has also claimed that the walls of a vessel can be seen when it crosses a dark background, such as is formed by another vessel.

When the blood stream is absent, the vessels are easily to be seen. Schnabel and Sachs state that after death the vessels in the papilla appear as light threads. In spontaneous and artificially produced arterial and venous pulsation the empty vessel walls are visible as bright bands, but this appearance must be differentiated from that caused by arterio-sclerotic thickenings of the walls, and also from that caused by the filling of the vessel with plasma, both of which produce the appearance of round cords.

Diseases of the vessel walls make themselves visible chiefly in two ways, as white sheaths from perivasculitis, and as more or less irregular constrictions of the calibre of the blood column, in which the etiological thickening of the wall cannot always be recognized with certainty. A uniform narrowing of calibre is found chiefly in chronic degenerative processes, particularly in retinitis pigmentosa. In these cases, with a plane mirror and reduced illumination the walls can often be seen as abnormally thick, light gray stripes on each side of the blood column.

The ophthalmoscopically visible changes from arterio- and phlebo-sclerosis have been well described by Raehlmann. The symptoms which interest us the most are those dependent on proliferations of the intima of the arteries, which can be recognized through constriction of the blood column while the outer contour of the vessel remains of its normal calibre. These constrictions are most distinctly to be

seen from the entrance of the vessels to about one papillary diameter into the retina, usually diminishing in intensity. How far they extend proximally can be determined only with the microscope, and very few such examinations have been made. Sometimes the thickenings of the walls are uniform for long distances, more frequently they extend for short distances and are not uniform. The blood column which has been constricted to a thin thread, often suddenly resumes its normal calibre, expanding so as to form a cone. When the normal places in the vessels are of short extent, peculiar spindles are formed which have often been mistaken for aneurysms. In other cases, the thickening of the walls extends in varying degrees for long distances, so that the blood column, diminished on the whole, has a peculiar irregular appearance, while more peripherally it is of normal calibre.

The thickenings of the walls are not always demonstrable. They are to be seen most easily with a plane mirror with reduced illumination, or by looking at the vessels in the margin of the cone of light, where they are to be seen by means of the light reflected from the posterior layer, when it appears as a gray or grayish-yellow cloudiness. In other cases, it has a bright gray, translucent, or grayish-white color and is very easily seen. In such cases, the thickening of the walls is very marked, so that that portion of the vessel appears as a solid, bloodless cord. But it has been demonstrated that blood can circulate through these places, and it is accepted as certain that it does do so when there is no stasis on the proximal side of the place in question. In regard to this, Raehlmann says that sometimes the affection is developed highly at the entrance of the vessels, in which case the principal arterial stems run into a gray mantle, from which they emerge suddenly and greatly thinned. The visibility of the thickening of the intima depends chiefly on the stage of development: it is transparent in the progressive stages, but becomes more opaque as the regressive changes progress.

The same vascular changes are observed in the so-called embolism of the retinal vessels, a fact which struck Raehlmann also, who says that one often finds the same condition

of the vessels after embolism of the central artery, and also that when years before he saw for the first time such a condition he thought he was looking at the remains of the embolism.

The arteries are described usually as much diminished in size, threadlike or invisible on the papilla and for a distance into the retina, and better filled in the peripheral parts. Irregular constrictions of the calibre have been described in some cases; more rarely the presence of a gray sheath as broad as the normal vessel, with a small central thread of blood, has been mentioned. This is partly because it is difficult to see these changes, partly because immediately after the attack the retina and papilla are rendered opaque by the more or less extensive necrosis of the nervous elements, and the vessels are thus hidden. If more attention is directed hereafter to these changes, they will be found more frequently, especially some time after the attack, when the opacity of the tissues has passed away. The limited localization and the form of these changes are sufficient evidence against the view of Elschnig that they are the expression of the nutritive disturbance of the vessel walls caused by anæmia, as well as Fischer's idea that they depend on the endothelial lining of the vessel wall.

Such arterio-sclerotic thickenings of the walls in embolism of the central artery have been described by many authors, but they have been found much more often in the so-called embolism of the branches, because the affected part lies in the clearer retina, and the contrast with the normal lumen is more striking. The vascular changes which take place when the circulation is first interrupted are followed after a time by secondary conditions which are in part due to an extension of the arterio-sclerosis and in part to a uniform diminution of calibre from functional adaptation, such as is seen in atrophy of other organs.

The tapering off of the veins, which is often conspicuous, may be due either to a phlebo-sclerosis, or, when it occurs in the papilla, it may be a normal appearance caused by the oblique passage of the vessel into the substance of the papilla. This appearance is often found associated with a

venous pulse at the entrance of the vessels, which is due to the same anatomically inclined position.

As has just been said, there are only three possibilities which can produce this clinical picture—embolism, thrombosis, and endarteritis proliferans, and as positive proof cannot be brought forward in favor of either one it is necessary to consider which can best explain all the symptoms.

The theory that thrombosis of the central artery is the cause of the interruption of the circulation was propounded by English authors and has found adherents in England and America. A clear differentiation must be made between the benign thrombus which follows a spontaneous degenerative change in the wall, and the toxic thrombus which appears in vessels in inflamed tissues. The latter certainly causes the sudden blindness in orbital phlegmon, with fundus changes which resemble the clinical picture of embolism, but this condition can easily be differentiated by the presence of other symptoms. The benign form of thrombus may be due to a degenerative disease of the inner wall of the vessel which causes loss of endothelium, or to a retardation of the blood stream. But while arterio-sclerosis of the larger vessels involves all the layers of the walls and destroys more or less of the inner wall so as to make a rough surface favorable for the formation of a thrombus, in the smaller arteries it usually takes the form of endarteritis proliferans, which is less apt to destroy the endothelium. This accords also with the nutritive conditions of the vessel walls, which are more favorable in the small vessels than in the large. But if no abrasion of the endothelium is present the formation of a thrombus will be difficult, particularly as the blood will be driven with increased swiftness through the narrowed place. Cohnheim says that it is an undoubted fact in human pathology that thrombi are never formed in vessels under a certain size, not even under conditions which favor coagulation of the blood during life and have at the same time caused them in larger vessels. How then does the clinical picture agree with the theory of thrombosis?

I agree with Mauthner that a complete interruption of the circulation takes place and lasts for a shorter or longer

time, in order to cause the sudden blindness and the partial necrosis of the retina from ischæmia. I can add to the reasons given by him that sometimes in cases of so-called embolism a slight improvement, or at least no further impairment, of vision is observed when the blood supply is very slight, as shown by the presence of a granular current, or by the very small calibre of the threads of blood enclosed in gray mantles which form the papillary or retinal arteries. Thus Raehlmann's case of great arterio-sclerotic thickening of the walls of the retinal arteries without defect in the visual field shows that a very small supply of blood will suffice to preserve the function of the retina. But it is certain that in almost all cases circulation is restored soon after the attack. If now the closure was thrombotic, there would be the greatest probability that the thrombus would extend proximally and distally, would become firmer, and would cause permanent total blindness with stasis in the retinal vessels. To explain how the circulation can be restored so soon after an attack, one must assume the theory of a canalization of the thrombus, or that of the formation of a collateral circulation. Canalization needs some time for its development so that total blindness would result in spite of it, and stasis would be met with because many cases are seen at an early stage. The possibility of the formation of a collateral circulation has been denied by competent investigators. The retinal vascular system is so isolated embryologically within the papilla that communication with the choroidal vessels is out of the question. It can then be formed only by the anastomosis of the branches of the central artery with Zinn's vascular circle. But Leber states that the anastomosing vessels are of very small size, little if any larger than capillaries. Collateral circulation could be developed but slowly through vessels of such size, while in the so-called embolism the vessels are pretty well filled again in a short time. The presence of the cilio-retinal vessels and of branches given off very early from the central artery is unimportant in this connection, because the retinal arteries do not anastomose.

How can the binocular attacks of different severity, the

prodromata, or the attacks which pass off without serious damage be explained by the theory of thrombosis? The cases in which a very slow granular blood stream can be seen coming out of the central artery, and in which a very small remnant of the lumen can be seen to be present, are particularly difficult to understand. It must be assumed that if there is a little canal remaining in the thrombus it will soon be stopped up by coagulated blood clinging to its rough surface. The impossibility of the theory of canalization is to be seen most clearly in those cases in which soon after the attack the blood column is greatly constricted for shorter or longer distances. Equally obscure are the cases in which for days at a time a granular current will alternate with an uninterrupted blood column, which shows that the movement of the blood is at times slower, at times faster, while the lumen at the place of obstruction is sometimes narrower than at others. Were the arterio-sclerotic thickenings of the walls which were seen in many cases purely coincidental? If this theory is right, a gradual change in the color of the thrombus from dark red to gray should sometimes have been observed.

The view which has been hitherto generally accepted is that the interruption of the circulation is due to embolism. But there is the same great difficulty in the explanation of the re-establishment of the circulation so soon after the attack. As total interruption of the blood stream must follow an ordinary embolism, and as this is not usually the case, the theory of an incomplete embolism has been propounded. According to this, the corners of an embolus of irregular form impinge on the walls of the vessel and the blood rushes by its margins, instead of its being driven forward by the force of the blood stream, until it completely fills the lumen. I think that such an embolus, which can be conceived of only as calcareous, is very rare. Emboli are formed from pieces broken from thrombi or atheromatous ulcers, both of which possess a fairly firm consistence. But as to the physical retention of a fissured body in the strong current of the vascular system, I believe with Mauthner and Leber that it will be driven as far forward as possible by the

strong current, that when the lumen becomes too narrow it becomes wedged in, and that its unevenesses are readily pressed into the distensible vessel walls. The only exception to this rule is when an embolus comes to rest at the point of division of a vessel, but even then it will usually close one of the branches. Elschnig tacitly admits this in his theory that where the embolus grazed the vessel wall a contraction of the latter was occasioned, which held it fast. It would then adhere so firmly to the intima that when the contraction was relaxed it would remain attached to the wall and allow the blood to again circulate. But unless it remains permanently fixed at this place the blood stream will crumble and loosen it and drive it farther on. If this theory is accepted as possible, it must be expected that as soon as the lumen widens a little the embolus will be carried forward in a mass until it is arrested in another place. I believe it to be impossible that an embolus can remain fixed to the wall of an artery, unless arrested by a projection from the wall or by becoming wedged in the mouth of a little branch. At the very first an embolus might turn, but this would allow the passage of a little blood for only a few seconds, because it would be driven at once as far forward as possible. Leber adds that increased obstruction through the formation of a thrombus, for which the rough surface of the embolus gives abundant opportunity, is to be expected rather than the opposite, a view with which Schnabel and Sachs coincide. The possibilities suggested by von Wagenmann, that the obstruction may be reduced in size by compression on the part of the vessel, or by a contraction on its own part so as to free a part of the lumen, or that the vessel may become dilated as the result of ischæmic paralysis, can bring about only the same conditions as would result from a relaxation of a contraction of the walls. A breaking up into minute particles, as suggested by Fischer, is scarcely conceivable on account of the general nature of emboli. If such particles should be unable to pass through the capillaries, they would cause many emboli of the smaller branches which could be plainly seen. Mauthner tried to explain the cases he met with by the theory that an embolus which

partly occluded the commencement of the central artery of the retina at the place of its separation from the ophthalmic artery was torn away after a short time by the blood stream. If this can occur, it must be with the greatest rarity. The rest of the symptoms remain unexplained, the same as with thrombosis, and Kern has shown that the source whence the embolus comes often cannot be demonstrated.

Among so many cases of this kind as have been observed, it would be a natural expectation that the embolus would have been seen frequently, but scarcely one of the pictures of supposed emboli can stand critical examination.

(Abstracts of the ophthalmoscopical appearances as described by different authors in twenty-two cases are omitted here from lack of space.)

Criticism of the descriptions given by various writers is difficult and uncertain, partly because they are brief and inexact, partly, perhaps, because they are subjectively colored by the desire to see the embolus. Greater clearness can be obtained in the future if more attention is directed to the condition of the vascular walls, especially if careful differentiation is made between ischæmic diminution, filling of the normal calibre with plasma, and arterio-sclerotic changes in the walls, the last of which may vary from a simple narrowing of the calibre of the vessel, with a not always demonstrable thickening of the walls, to its metamorphosis into a white cord of corresponding calibre, in the midst of which a canal may exist without any red blood being visible. In several cases the supposed emboli are described as having the appearance of white scales resting on the arteries, through which sometimes the red blood column could be seen. Whether these white spots were indicative of arterio-sclerosis, small extravasations, or membranes of connective tissue, cannot be told, but at any rate they were not emboli. In a few cases, short swellings of the arteries were pronounced emboli, while the vessel more centrally or more peripherally was pictured as more or less thinned. It is conceivable that these swellings were ectasiæ, or perhaps places of normal calibre in vessels whose walls were for the most part thickened by arterio-sclerosis. It is difficult to

understand how the fundus regains its normal appearance after a short time. Mules asks in regard to his case: "Why was the clot dead white and so easily dispersed? Where did it go?" In another case, the superior and inferior arteries of the papilla and the commencement of their branches are described as uniformly filled with a mass the distal ends of which were not sharply defined, and yet there was no stasis, and amaurosis was incomplete. Finally there remained of this mass only a long gray thread in the superior, and a similar one in the inferior, temporal artery. It is impossible that a fibrinous coagulum or an atheromatous mass should form so uniform an effusion, produce no stasis, and result in incomplete amaurosis. In one case, the statement that the artery was bloodless for a distance behind the embolus, awakens doubt as to the accuracy of the observation. Was arterio-sclerosis with the lumen of the vessel filled with plasma mistaken for bloodlessness? If so, perhaps the dark red plug was only the sign of a narrowed place in the vessel. After seven days, the embolus had changed its position. In other cases, the description is indicative of embolism, but too brief to be convincing.

In the picture of an embolus fixed in a vessel, one would expect to see it enclosed both distally and proximally by two points from the blood column, which would differ from it somewhat in color at least. We should also expect to see peripherally the blood column broken up into granules, but such a picture is not to be found among all the descriptions of supposed emboli. On the contrary, there has often been described a conical pointing of the blood column at each end of the place of obstruction, a condition which indicates a narrowing of the lumen through thickening of the walls.

(The reader is now referred to the writings of eighteen authors with regard to the conditions found by them microscopically.)

It is difficult to criticise these writings, because several of the observations were made before microscopical technique and histology had attained the modern standard, and because even at the present time certain pathological questions of importance in this connection have not yet been satisfac-

torily settled. Perhaps the differential diagnosis between an organized embolus, thrombus, and primary endarteritis proliferans cannot be made always with certainty, particularly in the later stages. All layers of the walls take part in the organization of emboli or thrombi in the large vessels, and there are always present vessels which grow out from the vasa vasorum through the adventitia and muscularis into the foreign body. Yet it might be possible, though improbable, that in the small vessels which have no vasa vasorum organization could take place from the intima alone, the adventitia and muscularis remaining intact, and in that case the difficulty of differentiation between primary and secondary endarteritis would be much greater. But we certainly have the right to exclude cases in which changes in the entire wall, not simply of the intima, as well as changes of the foreign body have not been described, although sufficient time for their development had elapsed between the attack and the enucleation of the eye, as not having been satisfactorily observed.

In some cases no emboli were found, but this does not prove the absence of such a lesion, because, as Hirschberg pointed out, the entire course of a vessel can never be accurately examined. I cannot believe that an embolus and its organized mass can completely disappear and leave no trace, as Elschnig suggests, and in two of the cases at any rate the time was much too short for this to occur. But it is well known that endarteritis proliferans often is confined to short distances.

Certain other cases have to be excluded from consideration because of lack of histological unity, non-recognition of post-mortem changes, or too brief and imperfect descriptions. In some cases it is questionable whether finely granular unorganized masses in the vessels, not associated with lesions of the intima or endothelium, were not post-mortem coagula rather than emboli, while in some the thickenings of the vascular walls suggest endarteritis proliferans. Priestley Smith and Ridley describe cases in which the lumen of the central artery of the retina was filled with an organized mass, the fibrillæ of which were arranged con-

centrically. Neither mentions any further change in the other layers of the walls of the vessels. This fact, together with the concentric arrangement of the fibres, renders the diagnosis of endarteritis proliferans or obliterans the more probable. The similarity of the concentric arrangement to that of blood clots, which Ridley cites, is only superficial; as the organization of a thrombus, or of an embolus, takes place more or less irregularly from the periphery, the extension of the tissue will assume a more radiating and therefore more irregular character. The fact that in these cases perception of light was present up to the last argues against thrombosis and in favor of endarteritis proliferans. Ridley himself says that the changes in the walls of the vessels were characteristic of endarteritis obliterans rather than of atheroma. The patient had renal disease also, which he thought to be the cause of the vascular trouble, but I believe it to be more probable that the renal disease was the result of the arterio-sclerosis.

Schnabel and Sachs found what they took to be an embolus adherent to the wall, but the description—"A hyaline, partly granular plug, without cellular elements, covered with two or three layers of epithelium"—shows a very slightly developed organization for an embolus three months old, and is not convincing that the condition was one of embolism.

Neither the description nor the illustrations of Elschnig's case indicate positively that it was one of an embolus six weeks old clinging to the wall of the artery. In one drawing, the intima is thickened in its entire circumference, but much more so on one side, which was taken to be the seat of the embolus. Elschnig thought that the swelling of the intima had filled out the angles of the embolus, but this does not account for the thickening of the opposite wall. At places, a certain concentric arrangement of the tissues can be seen. In the other drawing, the endothelium seems to have been sketched in schematically, but even so the picture of the superior papillary artery suggests something else than an embolus. It is an important fact that Elschnig found more or less arterio-sclerosis in the retinal arteries, some

branches obliterated, others with their lumen narrowed to small slits, others showing only a little thickening of the intima, these changes frequently extending for short distances. He thought the changes were due to interference with the nutrition of the vascular walls through the temporary ischæmia; but with so great a difference in magnitude, and their usual confinement to short distances after such a brief lapse of time, they may be interpreted with greater propriety as evidences of a general primary arterio-sclerosis.

With more certainty, I believe Wagenmann's case to have been one of endarteritis proliferans. This seems to me to be indicated both by the illustration, with the concentric arrangement of the tissue elements, and by the description. He says that a little slit-shaped lumen could be seen lying not only excentrically along the wall (unilateral proliferation of the intima), but also in places within the embolus (concentric proliferation). The lumen was surrounded by many layers of endothelium. If the vessel was followed from its entrance in the optic nerve, it could be seen to be considerably narrowed by the concentric proliferation of the cells of the intima up to the site of the embolus. The same condition was found peripherally from the embolus. The apparent dilatation of the arteries, which he mentions, may be ascribed to the fact that, where the arterio-sclerotic thickenings had taken place, the walls of the vessels could not contract *post mortem* as much as in the uninvolved portions. Wagenmann also found in all the retinal arteries more or less marked proliferation of the intima, sufficient to obliterate some of the small branches, which, as in Elschnig's case, was of arterio-sclerotic nature.

The description given by Nuel might be reckoned as one of an embolus in the process of organization, but it is striking that the second subdivision of the central artery is so much narrowed in the papilla and for some distance into the retina that its lumen sometimes seems to be wanting. In the retinal arteries also the lumen is much narrowed by thickening of the walls, evidently endarteritis proliferans. The description that there was a proliferation of the endothelium of the central vein, while the blood column which

filled the vein was divided into islets, is not clear to me. It suggests a thrombus in process of organization, but Nuel states that no thrombus was present. Yet under these circumstances the circulation which was present in the retinal vessels and caused numerous hemorrhages and venous stasis cannot be understood. Nuel ascribed it to a collateral circulation through the capillaries, as he could find no larger vessels through which it might be formed. Marple describes the embolus examined by him as a hyaline body which is stained by hæmatoxyline, contains some granules, —which may perhaps have wandered in,—and can be plainly distinguished from the wall. Red blood corpuscles lay distally and proximally. The intima was hypertrophied, the nuclei multiplied. Sections of the optic nerve showed that the walls of the central artery were thickened proximally through proliferation of the intima, which increased more and more until the vessel was completely filled. At this place the diameter of the vessel was greater than in normal places. Farther back the proliferation of the intima gradually decreased. Marple took this mass to be an organized thrombus, though he freely says it was difficult to distinguish the hypertrophic intima from the tissue of the thrombus. I think there are several grounds on which to base the belief that this was a case of endarteritis proliferans: the gradual increase in the thickening of the intima both distally and proximally, the absence of blood pigment so soon after the formation of a thrombus, the absence of reactive changes in the muscularis and adventitia, the concentric arrangement of the tissue, and the long slit-shaped space in the mass, which could not be a newly formed vessel, but was much more probably the remains of the lumen left by the endarteritic thickening of the walls. If the more distal structure was an embolus, it is strange that the thrombus did not attach itself thereto, as it furnished a most favorable opportunity, but I do not think it was an embolus. I think it was something that had no causal relation to the blindness,—perhaps a thick clot of blood plasma with some leucocytes,—for it cannot be conceived that the secondary thrombus should become so highly organized, while the embolus should

experience no change at all and cause no irritation on the part of the intima of the vessels, as the proliferation of the intima and the multiplication of the nuclei at this place appear to be analogous to the spontaneous thickening of the intima found farther back. Even if one supposes it to have been a chalky embolus, it must at least have become covered, not to mention that chalky masses cannot usually be cut without trouble with the microtome, and one would hardly expect that a chalky body, which is very hard, would be carried so high in the vascular system.

The case of retinitis hemorrhagica formerly described by me, and, as I think, one of Wagenmann's cases, were instances of similar temporary occlusion of the central artery of the retina by endarteritis proliferans. In an eye which had been blinded two months before by hemorrhagic retinitis and had been enucleated on account of glaucoma, there was found marked thickening of the intima of the central artery of the retina, which had greatly narrowed its lumen. Small arteries and veins were obliterated in many places in the retina. The non-obstructive thrombus in the central vein, the histological nature of which is still doubtful, could hardly have caused the blindness.

If we now review each case, bearing in mind all the points hitherto discussed separately, we must admit that in not one are all demands so perfectly met as to form a convincing argument in favor of embolism or thrombosis.

Three years ago, I had an opportunity to observe a case of so-called embolism of a branch in which it was clearly evident, from the peculiar change in the artery at the place where the retinal opacity began, that the lesion was not an embolus, but a marked narrowing of the lumen of the vessel from thickening of its walls. In a case of hemorrhagic retinitis, which I examined microscopically, I found a high degree of endarteritis proliferans in the central artery of the retina, and this made clear the mechanism of the production of the symptoms of so-called embolism. I will now give, partially abbreviated, the clinical histories of three cases of so-called embolism, the third of which came under treatment only a short while ago.

1. Mrs. J., thirty-two years old, came under observation Feb. 22, 1896. Patient stated that on Feb. 18th a shadow suddenly appeared without external cause before the right eye in the inner and upper part of the field. She had had acute articular rheumatism twice and for some years had had palpitation of the heart after climbing stairs. Health otherwise good. Married eleven years; three healthy children.

The blood column of the right inferior temporal artery on the papilla suddenly passed through a short stretch of great narrowness, so that it could be seen only as a delicate red line which spread out in a conical form at each end. The normal outer contour of the wall was visible as a fine haziness against the red background. Pressure on the eye produced pulsation in the normal central portion of the artery, as well as in the nasal inferior artery, which branched off before the narrowing commenced, but not in its distal portion. Peripherally from the constriction the artery and its branches regained an almost normal calibre. In the region supplied by the inferior temporal artery was a typical cream-colored cloudiness of the retina. Scotoma in the upper-inner sector of the field. Lungs normal. No glandular swellings or eruption on the body. Over the apex of the heart a loud systolic murmur, the second sound accentuated. Cardiac dulness extended somewhat to the right. Apex beat strong.

Treatment.—Massage and sodium salicylate *gm* 0.5 twice a day.

Feb. 24th.—The blood column at the place of constriction appeared thicker. Pressure on the eye caused the artery to pulsate on the distal side of the constricted place.

Feb. 27th.—The constriction had become still more dilated.

Feb. 28th.—The constriction had almost disappeared.

March 13th.—There remained only a faint cloudiness of the affected portion of the retina with white shining dots. The formerly constricted portion of the inferior temporal artery appeared as good as normal, so that no trace of the disease could be seen aside from a greater glistening than normal of the wall on the nasal side. Fields, taken Feb. 22, March 3 and 18, showed a gradual diminution of the scotoma.

Aug. 27, 1898.—The inferior papillary artery was about half as thick as the superior papillary artery. Its nasal and temporal branches were for a distance smaller than the corresponding branches of the superior artery, but more peripherally they again

became normal. For a distance of three fourths of the diameter of the optic disc, at the place of the former constriction, the inferior temporal artery possessed a delicate, clear, silver-gray mantle of the same calibre as that of the column of blood in the corresponding part of the superior temporal artery, which faded away at each end. For about four diameters of the optic disc farther, the inferior temporal artery was smaller than would be expected, and smaller than the corresponding part of the superior temporal artery. The blood column did not exhibit its normal uniformity, but showed little, irregular, short bends in its contour, thus causing dark places. In the inverted image it was noticeable that this part was considerably less visible than the peripheral parts, which in the upright image appeared uniformly normal. In the central part of the formerly affected portion there was a very delicate, smoky haze of the retina, with a considerable number of very fine white dots.

Vision of each eye, $\frac{6}{6}$; with cylindric correction, $\frac{6}{6}$.

2. Mrs. B., forty-eight years old. Came under observation Jan. 11, 1898.

Several months ago a darkness came suddenly before the right eye. The vision improved again, but a more accurate statement cannot be made because the patient gave the matter no great attention. Yesterday evening everything again appeared to her as if the right eye was veiled. After going to bed she seemed to see in the darkness many golden rays with the right eye. On the previous day she had been much fatigued by a long walk. This morning she closed her left eye and found she could see nothing with the right. Has always been in good health, except that when fatigued she has often had palpitation and oppression of the heart. Two healthy children. Menses lately irregular and of long duration, probably climacteric. No symptoms of syphilis.

Uniform hypertrophy of the left ventricle, heart sounds clear except that when the patient is agitated there is a cracked first sound over the apex and left ventricle (Professor Eichhorst). No pathologically enlarged lymph glands, lungs normal, radial arteries uniformly rigid; pulse hard, 100. Urinalysis showed 0.75 per cent. albumin, no sugar, a few hyaline casts, epithelium, and leucocytes; sp. gr., 1.011, daily quantity about 2000*ccm*.

Both eyes externally normal, media clear. Vision of right, some perception of light to the extreme temporal side alone; of left, $\frac{6}{6}$.

Left eye normal. The veins were unusually branching on the

papilla and showed a remarkable physiological tortuosity, particularly two convolutions made by a branch of the superior papillary vein on the disc.

The right pupil was of normal size, reacted consensually in a normal manner, but did not react directly. The fundus showed the typical whitish opacity of the retina of the so-called embolism of the central artery, with radiating stripes about the disc and very dense in the region of the macula. In the midst of the latter appeared a red, round spot, the margins of which were sharply defined above, but not below, with the central normal reflex of the fovea. The vessels were sharply cut by the retinal opacity, into which many of them disappeared. The papilla was reddened, with margins which could be obscurely seen below and above but not on the nasal side, and with a central, funnel-shaped physiological excavation which extended to the temporal margin. The vessels on the papilla formed a picture of separate, short, dark red stumps, which was at first unintelligible, but later, when the cloudiness had subsided, were made out to be the superficial portions of an extraordinarily tortuous vein, the deeper portions of which were hidden by the opacity. This tortuosity of the superior papillary vein was physiological, corresponding to that of the single venous branch on the papilla of the left eye. In the upper temporal quadrant of the disc were several very fine tortuous vessels which extended a short distance into the retina, and some fine linear hemorrhages. The veins, to anticipate the clearing up of the short vascular stumps, could nearly all be traced to the entrance of the vessels, and all exhibited a centripetal granular current. This was swiftest and most finely granular, like flowing sand, in the inferior temporal vein, which was the largest, less swift in the median inferior vein, in which could be seen the separation of the blood into short red and yellow cylinders of variable size, with peculiar, sharp, irregularly notched lines of demarcation, resembling fracture lines of cast-iron. In the inferior nasal vein the movement was still slower. The current in the superior nasal vein was also slow. In the superior temporal vein it was so slow that long stretches of red and bright yellow color were formed, and the cylinders sometimes stood quite still, or pulsated back and forth, either standing still on the whole or gradually working toward the papilla. The rapidity of the movement in the veins varied somewhat within narrow limits.

The inferior temporal artery could be traced into the funnel-

shaped excavation where it became veiled. The inferior nasal artery first became visible about a papillary diameter from the margin of the optic disc. In both the blood columns were uninterrupted. The superior temporal artery appeared first about two papillary diameters from the margin of the disc, and in places its course could be traced as a bright band. More peripherally it showed a very slow centrifugal granular current. Sometimes for several seconds this vessel would contain only plasma for a distance of from one to two papillary diameters, so it looked like a bright yellow streak, and then would come a cylinder of agglutinated red blood corpuscles. In the superior nasal artery, which became visible about three papillary diameters from the margin of the papilla, there was a uniformly swift, centrifugal, granular current. About half a papillary diameter from the nasal margin of the papilla, a little artery emerged, which had a very irregular, greatly constricted calibre, through which ran a rapid, centrifugal, granular current. The smaller arterial branches, especially in the region of the macula, had a strikingly dark color, so they could not be distinguished from the venous branches. The constricted portions of the arteries, where the reflexes were lost, were also of a darker color than normal. The circulation was slower in the veins than in the corresponding arteries.

The peripheral portions of the vessels, which were perfectly visible, were only moderately smaller than would normally be expected from comparison with the corresponding vessels in the other eye. Quite to the temporal side there was a group of small hemorrhages. Downward and outward in the extreme periphery was a small atrophic choroidal patch.

Treatment.—Diet for nephritis. Tr. digit., ten drops t. i. d. Massage.

Jan. 12th.—In the inferior temporal vein was a fixed blood column, no granular current, and in the inferior nasal and median veins the granular current was more rapid. Otherwise the condition remained the same.

Jan. 13th.—The retinal opacity had increased and the central red spot formed a more striking contrast. The thickness of the retina could almost be determined by the parallactic motion in the boundary zone of the little white dots which by their aggregation formed the retinal opacity. At the same time it could be seen that the red of the central spot was situated quite deeply, and that it presented a fine golden speckling, which is normal

and is caused by the united action of the pigment epithelium and the capillaries of the choroid.

Jan. 14th.—In the retinal opacity below the macula, dark areas could be seen along the vessels, such as are described by Nettleship. A rapid granular current was still present in the inferior and superior nasal veins alone. In the superior temporal vein was a very slow current with great agglutination of the cellular elements, which sometimes stood still and sometimes pulsated back and forth.

In the superior temporal artery the current was very slow, but varied a little in swiftness. This artery divided about three papillary diameters from the margin of the optic disc, one branch going upward and outward, the other upward. In the more horizontal branch the current came to a standstill, then after a while a pulsating movement began, by which the cylinders were pushed gradually backward to the point of division, where they were drawn up the vertical branch in the stream of plasma which filled it for long distances. A singular but very clear picture was formed as the red cylinders which occupied the full diameter of the horizontal branch were broken into little pieces as soon as they emerged, were impelled in an arch toward the nasal wall, rebounded, were carried up the vertical branch, and gradually united to form cylinders which occupied the full diameter of the vessel, separated by cylinders of plasma. After a time, a normally directed very slow granular current recommenced in the horizontal branch, again it came to a standstill, and again turned backward. Exact observations as to the currents in the other vessels unfortunately could not be made, because the patient became tired, and after a long rest a slow granular current was present in both. Pressure on the eye caused the proximal parts of the veins to appear smaller, but did not induce either arterial or venous pulsation.

Jan. 15th.—There was a very slow current in the upper veins. In the horizontal branch of the superior temporal artery, the phenomenon of a backward flowing stream could be seen again.

Jan. 17th.—The dark areas in the retinal opacity described by Nettleship had disappeared. At some distance from the papilla, all of the arteries and veins seemed to be pretty well filled and no granular current could be seen. The superior temporal and nasal arteries, almost to the margin of the papilla, looked like small rather opaque bands of a bright color, which could be easily seen

against the red background, and contained fine central irregularly outlined threads of blood.

Jan 19th.—The retinal opacity had plainly decreased and contained little bright-yellow shining spots, principally about the macula (cholesterin crystals?). Outside of these were many white dots, perhaps products of disintegration. Between the papilla and the macula was a hemorrhage one third the size of the papilla. The superior and inferior nasal arteries had white borders to about one papillary diameter from the margin of the papilla. The superior nasal artery was composed, at its beginning, of markedly constricted sections separated by short somewhat wider ones.

Jan. 24th.—There now remained only a little area of white retinal opacity situated to the nasal side and nasally upward from the macula, while the rest of the formerly opaque retina appeared as if delicately veiled. The cholesterin crystals had become more numerous, the cherry-red spot paler. A little above the macula was a fresh hemorrhage. The papilla was clearer and showed the paleness of atrophy. It was now evident that what had hitherto appeared like stumps of vessels were parts of a very tortuous superior vein. The inferior and superior papillary arteries could be clearly seen as rather transparent grayish-white cords, which were thicker than the blood columns of the corresponding arteries of the left eye. Their solid cord-like form was well shown by parallactic movements with the upright image. In the outer half of their course on the papilla, fine central threads of blood, which became thicker more peripherally, could be seen. The inferior papillary artery divided into three branches, the first of which went off nasally in the form of a thin thread of blood, covered for about one diameter of the disc by a delicate gray mantle. The blood column in the temporal branch passed through a short thickening, in which another twig was given off downward, which was also much constricted by a thickening of the wall visible as a delicate gray mantle. The calibre was irregularly but much diminished for some distance, and then came a short spindle-shaped swelling, which presented the normal calibre, color, and central reflex of the vessel, then again a constricted portion, which broadened conically into the normal peripheral portion. About one and a half diameters of the disc from the margin of the papilla, a macular branch was given off, which first appeared as a yellowish-white cord with a scarcely discernible central thread of blood, but farther in it appeared normal.

Until after it had crossed the vein the superior temporal artery could be seen only obscurely as an irregularly attenuated red cord, bordered by a fine yellowish-white haze, which made the external contour of the thickened vessel wall almost plainly visible. More peripherally it was normal. The superior nasal artery formed for some distance an irregularly thick small red thread, with short spindle-shaped thickenings in places. The wall appeared as fine haze. Farther on the artery was normal. The median nasal artery had become considerably smaller, both in the constricted part at its beginning and in the formerly almost normal peripheral portion. About two papillary diameters from the margin of the papilla, it was a fine irregular red thread, invisible in places, with a hazy white border. The superior macular artery could now clearly be seen on the papilla and for about one papillary diameter from it as a thin grayish-white band, with a very fine central red thread of blood. More peripherally it was normal.

Vision, right eye, movements of the hand on the extreme temporal side at 20 *cm;* left eye, $\frac{6}{6}$.

Jan. 25th.—Condition the same.

Jan. 27th.—Very little haziness of the retina now remained; the cholesterin crystals had mostly, the white dots largely, disappeared. The veins had white margins, in part unilateral, when it seemed as if there was between the white margin and the blood column a very small clear interspace, otherwise their calibre was normal. If this interspace exists, does the opacity belong to the adventitia or to the lymph sheath?

Feb. 8th.—The retinal opacity had gone, but there still remained a diffuse fine gray veiling of the formerly opaque parts. In the macula there was perhaps more pigmentation than formerly, and the white dots were scattered about its neighborhood. The hemorrhages between the macula and the papilla had been absorbed. On this day, a week since the patient was last seen, a granular current could be seen again in all the vessels. In both the superior arteries, the current was very slow, with long stretches of plasma and short cylinders of red blood corpuscles. In the horizontal branch of the superior temporal artery, the phenomenon described above could be seen again. In the afternoon, the blood in the horizontal branch stood still for some time and then began slowly to flow centrifugally. The superior temporal vein looked like a grayish yellow band in which the sparse masses of agglutinated red blood corpuscles could be seen to sink slowly.

The patient could give no reason to account for this condition except that on the previous day she became very tired. She had been feeling very well. Menses five days ago. Ordered rest.

Feb. 9th.—Same condition as on Jan. 27th. No granular current. Pressure on the eye caused no arterial or venous pulse, but did diminish the proximal segments of the veins and even cause them to disappear, although it could not suppress the threads of blood in the arteries as long as the pressure was not greater than the patient could bear. In the left eye a hemorrhage was to be seen external to and below the papilla.

Feb. 26th.—Papilla more markedly pale and atrophic. The thread of blood in the inferior papillary artery broader and extended farther proximally.

March 11th.—In the left eye, there were seen a small fresh hemorrhage and two little silver-white spots with radiating margins a little below and between the macula and papilla. There was an irregular constriction of the blood column of the superior temporal artery for some distance pretty nearly above the macula.

March 29th.—In the left eye, the hemorrhage and one of the white spots had disappeared, while the other spot had become smaller.

In the right eye, there was a steam-like haziness of the retina over the region of the posterior pole and the macula was very finely pigmented. Both veins and arteries, but particularly the latter, were smaller than they should have been in their otherwise normal peripheral sections. The white margins of the veins, usually unilateral in places, extended from three to five papillary diameters from the margin of the disc and were more marked than formerly. The proximal sections of the arteries showed an increased thickness of the threads of blood, and the junction of both papillary arteries with the central artery could be seen. The superior temporal artery was almost invisible for about one papillary diameter on the other side of the macula, but more peripherally it again became normal. No arterial or venous pulse could be obtained by pressure, but a very hard pressure caused a slow granular current.

Vision, right, amaurosis; left, $\frac{6}{6}$.

3. Mrs. H., sixty-four years old, came under observation July 1, 1898.

Patient had never been really sick, has borne six healthy children, and had no miscarriages. For the past three years has

often had swollen feet and eyelids, but these swellings went away of themselves after a little time. For the past three months her feet have been almost constantly swollen. She has often had palpitation of the heart after climbing stairs or other severe exertion, and has had frequent headaches since childhood.

Twelve days ago the patient noticed a shadow before the right eye for the entire day, which was worse on the following day. Eight days ago she accidentally learned that she could not see with the right eye, and the condition has remained the same ever since. She has not lately made any severe exertions.

Very pale, weakly woman. Radial and temporal arteries very rigid. Heart sounds clear. No cardiac hypertrophy. Pulse 90 to 100. Fairly marked œdema of feet and ankles. Eyelids slightly œdematous. Skin lax. Urinalysis: sp. gr., 1.009; daily quantity about 1100*ccm.*; albumin, 1 %; a few hyaline casts, some round cells and pavement epithelium.

Both eyes were externally normal, the media clear.

Right eye. Over the entire region of the posterior pole there was an irregular yellowish-white opacity of the retina with radiating marks about the hazy papilla. The red spot in the macula did not present a strong contrast because the retinal opacity had begun to retrograde. The formation of the opacity by the coalescence of little white dots could be recognized in the boundary zone opposite the central red spot, by observation of the parallactic motion of the dots on the more deeply lying red of the choroid. Between the macula and papilla were large thin irregular hemorrhages.

On the papilla the arteries resembled white cords with very fine threads of blood in the middle, visible only in the outer two thirds. As slightly larger threads, they extended for two or three papillary diameters into the retina and then their calibre became increased. Some short portions of the superior artery and its branches on the papilla remained normal, or were only slightly constricted, and gave rise to the appearance of short spindles. The thickened vessel wall could be seen as a gray mantle most plainly on the papilla, peripherally it became obscure and resembled a light border which was less red than the rest of the fundus. The inferior vein on the papilla and the beginnings of its branches were much constricted to about two papillary diameters into the retina. The blood column of the superior papillary vein was less constricted, but there was a marked constriction which affected

the blood column of its nasal branch somewhat more peripherally for the distance of about one and a half papillary diameters. Where the calibre of the veins was constricted, the blood column was enclosed in a gray sheath of rather greater thickness than would be expected in normal vessels, clearer and whiter than those of the arteries. In both arteries and veins, where the sheaths passed over into the normal contour of the vessels, the blood columns expanded conically. The calibre of the peripheral portions was only a little less than that of the corresponding vessels in the left eye. The inferior and superior temporal veins were much constricted where they crossed the arteries, without any thickening of the wall being visible. A granular current was present in all vessels, but no venous or arterial pulse could be obtained by pressure.

Left eye: The papilla was slightly reddened. There were some clear white spots, with diffusely radiated margins, about three fourths the size of the papilla, and some small hemorrhages in the fundus. The vessels were normal.

Vision, right, movements of the hand at 3*cm;* left, $\frac{1}{8}$.

Treatment.—Milk diet, rest in bed.

July 3d.—No granular current.

July 29th.—General condition better. Urinalysis, 0.75 % albumin.

In the right eye, there remains only a little opacity of the retina in spots, mainly about the macula. The vessels were in about the same condition as before. Pressure on the eye produced no arterial or venous pulse, but did cause a granular current. The conditions present could not be studied with exactitude because the patient was very weak and irritable.

I have only a few words to add to these clinical histories. The quick retrogression of the obstruction is most striking in Case 1. It was evident that the obstruction was not an embolus, from the conically pointed blood column on each side, together with the normal outer contour of the walls. That it was not a thrombus is shown by the persistence of a central canal, as well as by the rapid and uniform retrogression, which in endarteritis proliferans can be understood, if it is assumed that the proliferation came from the posterior wall. In that case, after the retrogression the diameter of the canal would appear normal, although a constriction might

exist in the diameter perpendicular to the retina without being visible on account of its position.

That a retrogression of the endarteritic proliferation can take place is shown clearly in both of the arteries on the papilla in Case 2. At first only a fine thread of blood was visible in the outer part of the course of each, but later this thread became thicker and was visible to its junction with the central artery. The fact that a blood current was always present shows that an extremely small thread of blood existed all the time, but it could become visible only when the thickening of the wall retrogressed. In the same way, the threads of blood in the previously constricted proximal sections of the retinal arteries became thicker and retained a whitish margin which grew clearer. This was not a counterfeit appearance caused by the dwindling of the retinal cloudiness, because the retina had already lost its white opacity. It is of the greatest importance that there should be stated from accurate observation of more cases of this kind the appearances present at any given time, together with exact delineations of them in regular epochs, as it is easy to err when the memory is relied upon, especially when such marked changes appear within a short time.

The fact that it was difficult to obtain a granular current in Case 2 by pressure on the eye indicates that the artery was very rigid, so it could not be readily compressed.

Hirschberg, Perles, Elschnig, and Uhthoff have also observed a turning backward of a granular current. Elschnig, who noticed that sometimes the blood ran backward from a branch into the main artery and then would be driven into another branch while the main stem again became white, ascribed this to a slow vermiform contraction of the main artery. Such an occurrence seems to me to be incredible, and I explain the phenomenon in this way. The agglutinated masses of red blood corpuscles sink in the plasma while it is standing still, and, when a little current of plasma is allowed to enter, are stirred up and come into view. The description given favors a filling of the vessel with plasma rather than a contraction. In a vermiform contraction a changing constriction of both the blood column and of the

vessel wall should be able to be seen. Part of the description is as follows: "In the glistening white superior nasal artery little blood cylinders slowly advance, . . . suddenly there is a complete standstill, the superior nasal artery becomes bloodless, while in its two branches the cylinders advance slowly. Shortly there is a standstill here also, then a backward current begins from the horizontal into the vertical branch, in which the current continues to run in the proper direction." Three hours later the central artery pulsated. The constriction must have been situated peripherally to the place of pulsation, but a satisfactory explanation is impossible because no note was made of certain points the importance of which this paper is to emphasize. I agree with Elschnig that the backward current is due probably to an influx of venous blood from a region in which there is a higher arterial pressure, for an anastomosis with other ciliary vessels cannot be supposed.

The white spots and hemorrhages which appeared in the opposite eye in Cases 2 and 3, as well as the associated nephritis, were results of the arterio-sclerosis. In Case 2 phlebo-sclerosis was also present. The very marked constrictions of the veins show to how great an extent the lumen may be diminished without causing stasis, tortuosity, or hemorrhages.

The mechanism of the production of the clinical picture of the so-called embolism is easily understood after a study of the microscopical examination in my case of hemorrhagic retinitis. The proliferation of the intima narrows the lumen of the artery more and more, the blood is forced through the constriction as long as its pressure is greater than the elastic contractile power of the arterial wall, so that the function of the retina is not interfered with. But a sudden change occurs if either the blood pressure sinks from any cause whatever, or if the tone of the muscularis is increased. In either case the walls will be brought together, particularly when the lumen is slit-shaped, the blood stream interrupted, and the function of the retina arrested. After a longer or shorter time the blood pressure will increase, or the contraction of the vessel walls relax. The damage to the ganglion

cells and nerve-fibres of the retina will be in proportion to the duration of the interruption of the circulation, and on this will depend the injury to the vision. After a very brief anæmia, the nerve elements can recover completely and the vision return to a degree approaching the normal, while the longer the anæmia continues the more will the nerve elements become necrotic and the greater will be the loss of vision.

Thus also is the fact explained that soon after the attack circulation is re-established in the retinal vessels. That the fulness of the vessels increases for a time may be explained by the retrogression of the endarteritic proliferation, which also is perhaps a result of the temporary ischæmia.

Such prodromal attacks as those described by Schnabel and Sachs can also be understood as the direct result of fluctuation in the balance between the tension of the arterial wall and the blood pressure. If some time intervenes as the result of regressive changes, which for a time render the lumen free, new proliferations of the intima occur in the neighboring parts of the walls and cause a fresh attack.

The bilateral attacks can also be understood. In such cases endarteritic changes are present in both central arteries, which cause longer or shorter interruptions of the circulation whenever the blood pressure is reduced, and thereby different degrees of injury to the two eyes.

The so-called transition of an embolism from the main trunk to a branch can also be thus explained. There is a marked thickening of the intima of the affected branch which does not yield when the blood courses again through the central artery, and thus greater disturbances are caused in the region supplied by it.

This theory is supported most of all by those cases in which a granular current is present for some time and then suddenly disappears. A condition in which an artery is filled for many seconds with plasma alone, and then shows a little cylinder of red blood corpuscles ascending, can be explained I think only by the theory of endarteritis proliferans. The cellular elements will glide over the smooth endothelium as long as there is possible room, while they

would have to cling to the rough surface of the thrombus or embolus and be brought to a full stop. Occasionally the cellular elements may accumulate in front of the place of obstruction, where the smallness of the slit allows only the passage of plasma, but as soon as the lumen enlarges, whether actively or passively, they will pass through again.

The cases of Hirschberg, Knapp, and Uhthoff, in which certain arterial branches were at first not involved, but were affected later, can be explained as due to the extension of the endarteritis proliferans.

The idea that the clinical picture of embolism of the central artery is caused by disease of the vessel walls has often been suggested, but the lesion has usually been assumed to be atheroma and consequent thrombosis. In a case of albuminuric retinitis, Hirschberg traced a defect in the visual field to closure of an artery by disease of its walls. Haab, in his observations regarding syphilitic arteritis, remarks that many cases of embolism, in which the source of the embolus is not evident, may perhaps depend on syphilitic endarteritis, and not long ago he advocated the theory of the endarteritic origin of the disease in question. In the same way, Scheffel's case may be thought to be due to syphilitic endophlebitis rather than to thrombosis. It is incomprehensible that long stretches of a vessel can be thrombosed and it still retain a small canal in its middle. At some time the thrombus must cause total closure of the vessel with the resultant symptoms, but this does not seem to have been the case. The rapid retrogression of the symptoms under anti-syphilitic treatment also favors the idea that the lesion was endarteritis proliferans—a direct result of syphilis, rather than a thrombus—a secondary result.

On the basis of a microscopical examination, Raehlmann explains the sudden bilateral blindness of a patient after confinement as due to endarteritis proliferans, and draws attention to the fact that such a condition would explain the hitherto obscure attacks of blindness after loss of blood, and it is very probable that a causal relation exists between such attacks of blindness and endarteritic change in the walls of the central artery of the retina.

A short time ago, Markow quoted the picture of embolism as analogous to that of endarteritis obliterans in spontaneous gangrene.

Heubner was the first to draw attention to the importance of endarteritis proliferans, particularly with regard to diseases of the brain. He believed it to be specifically syphilitic, but this is not the case. It may be caused by syphilis, but is usually a part of a general arterio-sclerosis—primary in that a direct cause has not yet been ascertained, except in some secondary forms which have been described under the name arteritis obliterans. The symptoms of endarteritis proliferans or obliterans have become known to us principally through the work of surgeons in the investigation of spontaneous gangrene. They have shown that the disease is widespread and occurs at all ages. The clinical picture of spontaneous gangrene much resembles that of embolism of the central artery, particularly in its manner of attack. That it frequently results in gangrene of the extremities, but causes necrosis of the nerve elements only in the eye, is in accordance with the peculiar nutritive conditions. In their more dependent position the extremities are more prone to stasis of blood, exposed to injury, and depend for their nourishment on the branches of one artery, while in the eye the lymph stream from the chorio-capillaris suffices for a part of the nutrition of the retina, so that when the central artery is closed the supporting substance of the retina retains its vitality, although the sensitive nervous elements are ruined. The retina is nourished by two systems of vessels in a measure independent of each other.

We do not yet know whether this disease finally results in obliteration of the retinal vessels or not, because the patients seldom remain under observation sufficiently long after they have become blind for this to be determined, but it is possible that after it has reached a certain degree the endarteritic proliferation may undergo a retrograde metamorphosis. This may have been the condition in the cases observed by Raehlmann and others, in which there was a marked constriction of the lumen from arterio-sclerosis, persisting in some of them for a long time, without total closure of the

vessel or stoppage of the circulation. I have seen in a woman forty-one years old, who had a glaucomatous excavation of the papilla, a short well-marked constriction of the lumen of a retinal artery near the margin of the optic disc, the peripheral portion of which retained very nearly its normal calibre, and in a man sixty-eight years old nearly all the branches of the central artery much constricted in places on the papilla and in the neighboring parts of the retina, while more peripherally their calibre was only moderately and uniformly reduced. In neither case was there a scotoma.

The source from which an embolus may have come can by no means always be demonstrated, but almost all of these patients have arterio-sclerosis. Even when the disease is not present in the arteries which can be palpated it may exist in other arteries, and many of the demonstrable defects of the cardiac valves wholly depend on arterio-sclerotic changes without thrombus formation, which furnish no material for an embolus. Arterio-sclerosis may be found simultaneously in the eye, kidneys, brain, coronary arteries, and other organs.

It can hardly be considered an accidental coincidence that arterio-sclerosis so often affects the central artery and its branches, and that changes similar to those produced by it have been described in so-called embolism, especially of the branches. It is more reasonable to think that endarteritis proliferans should be considered the chief factor in the production of the clinical picture in question, particularly as all the symptoms can thus be satisfactorily accounted for. Even if arterio-sclerotic changes should not be visible in the artery, they might be present proximally to the point where it comes into view.

Thrombosis and embolism can happen in the central artery or in one of its branches, but if the circulatory conditions in the eye are correctly understood, either lesion must cause a total and long-continued interruption of the circulation, visible signs of agglutination in the vessels, and complete blindness from the first. In embolism of a branch the embolus should be plainly seen.

I agree with the commonly accepted opinion that the

retinal opacity is caused by the granular haziness and swelling of the nervous elements of the innermost layer of the retina, together with a more or less marked serous exudation, a condition similar to the post-anæmic changes in the kidney, spleen, uterus, and other organs, which are known under the name of ischæmic or white infarction. In time the necrosed tissue elements are absorbed so that the white opacity disappears, but the retina does not seem to regain its normal transparency, a delicate rough haze remains, most plainly to be seen at the posterior pole.

Extensive retinal hemorrhages have been observed very seldom, perhaps because when they do occur soon after the attack the case is diagnosed as one of hemorrhagic retinitis. I believe that cases of sudden failure of vision which present the ophthalmoscopic picture of hemorrhagic retinitis with small arteries and few or no choked veins belong to this class. Why hemorrhages should occur in one case and not in another I do not know. No satisfactory explanation has yet been made of the so-called hemorrhagic infarct in general pathology, but it is known that they can be produced experimentally only under very favorable circumstances. In the eye various factors may need to be taken into account, such as the general condition of the vessels, the duration of the interruption of the circulation, the amount of injury to the vessel walls from the anæmia, the height of the blood pressure, and the condition of the blood itself.

In regard to the red spot in the macula, I adhere to the usual view that it is caused by the choroid showing through the unclouded retina at this point, and that its color appears intensified through contrast with the surrounding white opacity. In Case 2 a fine physiological marking of the choroid could be seen, and the thickness of the retina almost estimated from the parallactic movements of the white dots against the red of the chorio-capillaris and pigment epithelium. The color of the spot naturally varies with the amount of pigment in the pigment epithelium and choroid in individual cases. The microscopical changes in the pigment epithelium found by Elschnig may have been only

secondary, because it is difficult to understand how they could have been produced directly by the attack, and it is known that the pigment epithelium is secondarily involved in very different retinal diseases. But such changes cannot usually be marked in so-called embolism, because a fine pigment granulation, such as is sometimes seen in a senile macula, is often mentioned, especially of late, among the ophthalmoscopic appearances.

The optic atrophy which results from so-called embolism is in unison with the view held at the present time, that the optic-nerve fibres appertain to the ganglion cells of the retina and are involved in their atrophy.

Conclusions.—(1) There can be said to be complete interruption of the circulation only when the blood column is broken to pieces on the peripheral side of the place of obstruction.

(2) While the theory of embolism or thrombosis does not explain many of the symptoms of the clinical picture in question, the theory of endarteritis proliferans of the central artery, or of the retinal artery affected, satisfies all the demands which may be made upon it, and this disease is present at all events whenever thickenings of the walls of the arteries are visible with the ophthalmoscope. With true embolism or thrombosis, both of which may occur in the retinal arteries, complete blindness and a breaking up into granules of the blood columns of the affected retinal vessels must be expected.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FOURTH QUARTER OF THE YEAR 1902.

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WITH THE ASSISTANCE OF

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Translated by Dr. Ward A. Holden.

Sections I.–III. Reviewed by Prof. HORSTMANN, Berlin.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

461. **Groenouw** and **Uhthoff.** The relations of general and organic diseases to affections of the eye. *Graefe-Saemisch*, 2d edition, Leipsic, 1902.

462. **Bernheimer.** Etiology and pathological anatomy of ocular-muscle paralyses. *Ibid.*

463. **Hess.** Anomalies of refraction and accommodation with an introduction to the dioptrics of the eye. *Ibid.*

464. **Snellen.** Eye operations. *Ibid.*

465. **Lagrange** and **Valude.** *French Encyclopædia of Ophthalmology*, Vol. I. History of Ophthalmology. Anatomy of the Eye and its Adnexa. Physiology. Paris. O. Dorin, 1903.

466. **v. Reuss.** The visual field in functional nervous diseases. Leipsic and Vienna, 1902. F. Deuticke.

467. **Javal.** How to counsel adults who become blind. *Bull. de mém. de la soc. franç. d'opht.*, xix., p. 268.

468. **Eversbusch.** Practical testing of the color sense with the signal lights used in the railway service. *Deutsche med. Wochenschr.*, 1900, Lit. Beil., p. 302.

469. **Kubli.** Brief reports from eye practice. *Wjest. ophth.*, 1902, No. 6.

470. **Brüning.** Report on the work done in the University eye clinic at Tübingen from 1875 to 1901. Tübingen, 1902.

471. **Bock.** Twelfth report on the department for diseases of the eye in the hospital at Laibach.

472. **Wood, C. A.** Address on an exhibit of early (prior to 1860) British and American ophthalmic literature. *Journ. Amer. Med. Assoc.*, Nov. 8 and 15, 1902.

GROENOUW (461) describes metastatic ophthalmia, the affections of the eye in sepsis, splenic fever, actinomyces, trichinosis, etc. Further chapters treat of ocular affections in measles, scarlatina, smallpox, vaccinia, varicella, and erysipelas.

The article by BERNHEIMER (462) forms the conclusion of his excellent treatise on the etiology and pathological anatomy of the paralyses of the ocular muscles.

Arlt's treatise on operations in the first edition of *Graefe-Saemisch* has been worked over by SNELLEN (464), since in the last two decades more changes have occurred in this department than in any other. Following a section on anæsthesia, antisepsis and asepsis, mydriatics and miotics, instruments and dressings, he describes the operations for cataract, iridectomy, glaucoma operations, the operations for staphyloma of the cornea, enucleation, and exenteration.

LAGRANGE and VALUDE (465) have prepared with the assistance of colleagues of the Latin lands a French handbook of ophthalmology after the model of Graefe-Saemisch. The first of the projected eight volumes contains the following articles: Pansier, a short history of ophthalmology; Lagrange (Bordeaux), general anatomy of the orbit; Motais (Antwerp), anatomy and physiology of the motor apparatus of the eye; Rollet (Lyons), anatomy of the pneumatic cavities of the face; Terson (Paris), anatomy and physiology of the eyelids; Kalt (Paris), anatomy and physiology of the lachrymal apparatus; Berger (Paris), general anatomy of the eyeball; Morax (Paris), anatomy of the conjunctiva and cornea; Rohmer (Nancy), anatomy and physiology of the sclera; Venneman (Loewen), anatomy and physiology of the uveal tract; Truc and Vialleton (Montpellier), anatomy of the lens, with a historical introduction by Dor (Lyons); Rohmer

(Nancy), anatomy and physiology of the vitreous; Rochon-Duvignaud, anatomy of the sensory nervous apparatus of the eye: retina (under the direction of Mathias Duval), optic nerve, optic-nerve centres. Numerous illustrations, many of them new, accompany each article. The mechanical book-work is excellent.

BERGER.

v. REUSS (466) describes the visual field in functional nervous diseases. His material includes 85 cases, only 7 of which were not traumatic. Of these 78 cases, 59 followed railway accidents, 5 a fall on the ground or from a low elevation, and 2 a fall from a greater height; the rest followed different injuries.

He divides the cases into two groups: the earlier, 32 in number, seen before Nov., 1898, and the later 53 seen since that date. In the latter group fatigue symptoms were always looked for and found in 40 cases. In the general part of the monograph, v. Reuss describes the normal field of vision and then the defects such as are found in hysteria and neurasthenia, the displacement type, Wilbrandt's fatigue type, concentric contraction, the so-called fatigue-spirals, the rest extensions, and finally the mixed forms. The special part contains the 32 earlier cases, of which 13 had normal fields and the rest contractions. Among the 53 later cases fatigue-spirals were found in 40 cases or 70 eyes. Only 22 eyes had fields with normal limits. The ophthalmoscopic examination was almost invariably normal, and diminution of vision was found in but 25 persons in one or both eyes. The fatigue is no retinal process, but a psychical fatigue.

EVERSBUSCH (468) has had constructed a testing apparatus which furnishes an exact representation of the day and night signals. For the night tests he uses blue and yellow as well as green and red in his lantern, in order to render more difficult the orientation in respect to green and red, which the difference in luminosity assists in the color-blind.

From 1875 to 1901, 17,518 patients were admitted to the Tübingen University eye clinic (470), and 46,615 out-patients were treated. The number of cataract operations was 3546, and of enucleations, 845.

BOCK (471) in 1901 treated 1273 patients in the Laibach hospital: Cataract operations, 104; iridectomies, 103; squint operations, 6; and enucleations, 20. No suppuration after cataract operations.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

472 A. **Saffner.** Bulbus septatus. *Graefe's Archiv*, liv., 3, p. 509.

473. **Orloff.** On the eye changes in chronic poisoning with secale cornutum and its preparations. Preliminary communication. *Wratsch*, 1902, No. 53.

474. **Woitzechowsky.** On metastatic affections of the eye in general and local infections. *Wjest. ophth.*, 1902, 4–5.

475. **Bohn.** Congenital and acquired pathological pigmentation of the eyeball. *Inaug. Dissert.*, Giessen, 1902.

476. **Golowin.** On changes in intraocular tension from compression of the carotid. *Wjest. ophth.*, 1902, 4–5.

477. **Engelmann.** Tonometric investigations on healthy and diseased eyes. Juriew, 1902.

478. **Dimmer.** Photography of the fundus. *Berl. klin. Wochenschr.*, 1902, No. 49.

480. **Trantas.** Ophthalmoscopy of the ciliary body. *Ann. d'ocul.*, cxxviii. p. 301.

481. **Thorner.** Photography of the fundus. *Berl. klin. Wochenschr.*, 1902, No. 43.

482. **Hinshelwood.** Congenital word-blindness. *Ophth. Review*, April, 1902.

483. **Randolph, R. L.** The rôle of toxins in inflammations of the eye. (Prize essay.) *Amer. Journ. Med. Sciences*, Nov., 1902.

SAFFNER (472 A), on enucleating a child's eye that was larger than the other, lacked the lower segment of the iris, had an opacity of the corresponding portion of the cornea, and exhibited what appeared to be a cyst behind the cloudy lens, found a tract of tissue passing forward from the disc and ending in part about the lens and in part in the wall of a cystic cavity. This cavity was situated at the bottom of the ball between the margins of an iris and ciliary coloboma, and extended into the anterior chamber; the lens was luxated upward and inward. The tract of tissue was not connective tissue but glia tissue.

ORLOFF (473) used in his experiments chickens, rabbits, guinea-pigs, and cats—in all twenty-one animals. In four animals cataract developed. In many, mydriasis and almost complete loss of light reaction was observed. The microscopic examination of the retina revealed a vitreous degeneration of the vessels in some eyes and various changes in the ganglion cells up to their complete destruction. HIRSCHMANN.

BOHN (475) reports four cases of congenital melanosis of the sclera and two of the cornea. Acquired melanosis may be caused

by the healing in of powder grains or by the entrance of soluble coloring matters like anilin.

The manometric method of determining the intraocular tension is not applicable in man and is otherwise very uncertain because of the necessity of introducing a canula.

The results obtained by GOLOWIN (476) in normal eyes by this method, and also by compression of the carotid or jugular, and the ligation of the venæ vorticosæ; are too divergent to be regarded as accurate.

The tonometric method, the only one that can be used on man, is much more accurate. The best instruments are those of Fick and Maklakoff, the latter being, in the writer's opinion, preferable. Measurements made with this instrument on fifteen eyes before beginning the compression of the carotid, one-half minute, one minute, and two minutes after beginning the compression, and again after ceasing the compression, gave the following results: In a group of seven eyes the tension before compression was normal, but decreased from 1–5.5*mm* Hg (on an average 2.5*mm* Hg) during the compression. In five other eyes with glaucomatous increase in tension before compression, the tension was reduced a varying amount after compression was begun, but long continuance of the compression did not lead to further reduction but rather to increase. HIRSCHMANN.

The valuable treatise by ENGELMANN (477) contains chapters on the following topics:

1. A short account of the tonometry of the eye. Maklakoff's tonometer.
2. The influence of blood tension upon intraocular tension.
3. Intraocular tension in general narcosis.
4. The influence of the sympathetic upon intraocular tension (*a*) in animals, (*b*) in man.
5. What effects has the play of the pupil upon the height of the intraocular tension in man?
6. The influence of the extrinsic ocular muscles upon the intraocular tension.
7. Intraocular tension in accommodation.
8. Tonometric measurements in various diseases of the eye.

HIRSCHMANN.

TRANTAS (480) finds that if gentle pressure is made upon the ciliary region with the thumb after atropine and cocaine have been used the ciliary body may more readily be seen. The examination

is made in the upright image with a lens of 4–5 D. The writer presents drawings of the ophthalmoscopic picture of the ora serrata in the normal eye (of which he distinguished four types), in irregular atrophy of the pigment, in colloid excrescences, and in hereditary syphilis. BERGER.

THORNER (481) presents satisfactory photographs of the fundus of the cat taken by means of his ophthalmoscope, which eliminates the corneal reflex.

HINSHELWOOD (482) reports two further cases of this condition. One was in a girl aged ten, who was in all other respects quite healthy yet she had the greatest difficulty in learning to read. It took her nine months to learn the alphabet and after four years she could only read the simplest words without spelling them.

The second case was that of a boy aged seven, also in other respects quite healthy and particularly sharp, but he could scarcely read a single word without spelling it, though he could be taught easily enough orally, and he escaped detection for some time by learning his reading by heart. The first case has not been seen since, but the second after special reading lessons repeated frequently got on wonderfully well. It is useless to have such children educated in ordinary classes with others without this defect; they must be taught separately with lessons specially adapted to overcome their difficulty. The author is most emphatic on such children being examined by competent persons, otherwise the most useful years of education are lost and they become backward and defective through life. MARSHALL.

Infectious bacteria produce inflammation in the conjunctiva by their presence and by the production of toxins. The part played by the latter is the problem attacked by RANDOLPH (483). In a series of forty experiments on rabbits, the toxins from the gonococcus, staphylococcus aureus, Klebs-Löffler bacillus, bacillus coli communis, micrococcus epidermis albus, and xerosis bacillus were kept in contact with the conjunctiva constantly, often for many hours, and in practically every case no reaction followed. On the contrary, when these toxins were injected into the conjunctival tissue more or less violent reaction was produced, and when injected into the anterior chamber iritis was caused in every case.

Bearing on the subject under consideration, experiments were made to determine the presence of bacteria in the conjunctival sac of the healthy rabbit. These resulted in the conclusion that quite a variety of micro-organisms are normally present, as is the

case in the human conjunctiva. This point might have some influence on the results obtained from the injections, and the author believes that, although the toxins are directly responsible for the essential damage, as was shown by control experiments, they may, at the same time, impair the resisting power of the tissues to the bacteria already present. The foregoing experiments demonstrate that when the epithelium is intact and no opportunity for penetration is offered, toxins produce no local injury to the animal. It is suggested that the injection of bacterial infiltrates into the eye, particularly into the conjunctiva, constitutes a more delicate biological test for the detection of certain toxins than the methods usually employed. ALLING.

III.—INSTRUMENTS AND REMEDIES.

484. **Fuchs.** On cocaine. *Wiener klin. Wochenschr.*, 1902, No. 38.
485. **Kasass.** Eserine in keratitis. *Wjest. ophth.*, 1902, No. 6.
486. **Cervicek.** On the use of itrol and cuprocitrol. *Der Militärarzt*, 1992, 19–20.
487. **Kubli.** Ichthargan in eye practice. *Wratsch*, 1902, No. 49.
488. **Skorjuchew.** On the use of brucin in eye practice. *Inaug. Dissert.*, St. Petersburg, 1902.

In all operations, even the most trivial, FUCHS (484) instils a few drops of a 5 % cocaine solution, keeping the eyes closed in the intervals. If there is much injection he uses adrenalin 1:1000 or injects cocaine beneath the conjunctiva. In operations on the lids cocaine is injected beneath the skin, in muscle operations beneath the conjunctiva, in operations on the lachrymal passages into these. In chalazion he instils cocaine into the conjunctival sac and injects a drop or two into the chalazion itself. Schleich's infiltration method is not to be commended for operations on the lids and muscles, since the œdema renders the operation difficult.

KASASS (485) recommends, in all cases of keratitis, whether ulcerous or non-ulcerous, with the exception of parenchymatous keratitis, the use of eserin salicyl. in the form of a salve with or without xeroform, instead of atropine. In miliary phlyctenulæ also the effect is brilliant. HIRSCHMANN.

CERVICEK (486) treated one hundred and twenty-four cases of blennorrhoic conjunctivitis and eighteen cases of secreting trachoma with itrol. This is more irritating than a 2 % silver solution, but its effect lasts longer. He applies it with a powder blower, so that the action of light and air upon the very delicate

preparation is avoided. After using it a few times the irritability of the eyes is much diminished. He treated twenty-six cases of trachoma with cuprocitrol in various stages with good effects in part, using a 5-10 % salve and massaging the eye.

KUBLI (487) used 1-10 %, but mostly 2 % solutions of ichthargan. In twelve hundred patients he obtained the following results: In inflammation of the margin of the lid, if mild, ichthargan acted favorably; in severe cases, nitrate of silver is to be preferred. In cases of hordeolum, eczema, wounds, and abscesses, ichthargan favors recovery. In acute conjunctivitis, blennorrhœa, trachoma, and pannus, its effect is far inferior to that of silver or copper. It does not cause argyrosis.

SKORJUCHEW (488) concludes from many observations that brucin increases the central and peripheral vision in both healthy and diseased eyes, increases the strength of the muscles and diminishes their pareses, and frequently dilates the vessels, chiefly the arteries, of the fundus.

Brucin can be used with advantage in various amblyopias, in acute and chronic retrobulbar neuritis, optic-nerve atrophies, retinitis pigmentosa, and paralysis of accommodation. It is contra-indicated in acute inflammatory affections of the coats of the eye. HIRSCHMANN.

Sections IV.–VII. Reviewed by DR. ABELSDORFF, Berlin.

IV.—ANATOMY.

489. **Prokopenko.** On the distribution of elastic fibres in the human eye. *Graefe's Archiv*, lv., 1, p. 94.

490. **Howe, L.** A study of the connective tissue of the orbit by a new method. (Prize essay.) *Annals of Ophth.*, Oct., 1902.

PROKOPENKO'S (489) investigations, made chiefly by means of Weigert's and Unna's stains for elastic tissues, cover the distribution of the latter in all the tissues of the human eye, and in the main confirm the results of other recent investigations. It may be mentioned that the writer found elastic fibres in the cornea, but not in the iris; the limiting membrane beneath the pigment epithelium of the iris differing in its staining properties from elastic tissue, and being considered by him a dilatator muscular tissue.

HOWE (490) uses Zenker's fluid for fixing and alcohol for hardening, removing excess of mercury with Gram's solution. Then, wash in water; second, place in one per cent. solution of potass.

permanganate; third, wash; fourth, one and one half per cent. solution of oxalic acid until decolorized; fifth, wash; sixth, stain with fuchsin; seventh, wash; eighth, stain with anilin blue; ninth, wash; tenth, alcohol-xylol balsam. The method is similar to that of Mallory, but the use of permanganate and oxalic acid renders the tissues more susceptible to stain. The study of specimens of Tenon's capsule confirms the views of Schwalbe that, while the capsule is contracted at the nerve entrance, there is still a passage between the space around the globe and that about the nerve. The capsule proper is shown not to extend farther forward than the insertions of the muscles. Sections also show extensive attachments between the capsule and the muscles as well as to the soft parts and the edge of the orbit. The check ligaments, which are thickenings of the aponeurosis which passes from the edge of the orbit to the capsule and muscles, are fully described. Beside the internal and external check ligaments, the author designates others as the inferior and superior. Photographs of the connective tissue attached to the muscles and of the fascia orbito-ocularis add a new and helpful feature to methods of demonstrating these tissues.

ALLING.

V.—PHYSIOLOGY.

491. **Schoute.** The retinal cone in its function as end organ. *Zeitschr. f. Augenheilk.*, viii., p. 419, 1902.

492. **Reimann.** The apparent enlargement of the sun and moon on the horizon. *Zeitschr. f. Psychol. u. Physiol. d. Sinnesorgane*, 30, p. 1.

493. **Levinsohn, G.** On the relation between the cerebral cortex and the pupil. *Zeitschr. f. Augenheilk.*, viii., 5, p. 518.

SCHOUTE'S (491) investigations were toward the solution of the question whether the individual retinal cone as an end organ had only the function of conducting the intensity of the excitation—*i. e.*, only quantitative differences. The same amount of light falling upon a cone must then always give rise to the same perception.

Thus, in fact, different forms on a cone were always seen as round. It was possible further, by other experiments, to show that the area perceived with a cone did not depend upon the extent of the image, but only upon the amount of light. Since the latter is the product of the area of the image and its luminosity, a large image on a cone appears no larger than a small image of greater luminosity.

REIMANN (492) gives a historical résumé of the question in regard to the larger appearance of the heavenly bodies near the

horizon and offers the following explanation of his own: With the increasing tenuousness of the atmosphere above the refraction of light is weaker, and therefore much less diffuse light comes from the zenith than from the horizon. On the lower darker strata of atmosphere the light particles in the air are projected as an apparent sky, and therefore the sky at the zenith appears nearer than at the horizon. The visual angle remaining the same, objects near the horizon, which is apparently farther distant, appear larger than objects at the zenith.

LEVINSOHN'S (493) experiments were made on monkeys, cats, and dogs—those on monkeys being most satisfactory—after the sympathetic or the superior ganglion on one side had been extirpated. It was not possible to produce contraction of the pupil by the excitation of particular points in the cerebral vortex. A dilatation of the pupil was obtained, however, not only with the epileptoid convulsions caused by excitation of the cortex and the excitation of the motor sphere, but from three particular spots: (1), the cervical sphere, (2) the sensory sphere of the eye, and (3) the visual sphere. The dilatation usually took place in both eyes, but was less pronounced on the operated side. Since the dilatation rarely appeared alone, but was accompanied in the beginning by movements of the eyes or lids, and since extirpation of the cortical points in question caused no permanent symptoms, the dilatation of the pupil must be only a secondary symptom, attributable, in the writer's opinion, to simultaneous relaxation of the oculomotor and excitation of the sympathetic.

VI.—REFRACTION AND ACCOMMODATION.

494. **Einthoven.** The accommodation of the human eye. *Ergebnisse des Physiol.*, vol. i., Wiesbaden, 1902. Bergmann.

495. **Liebreich.** The treatment of myopia. *Klin. Monatsbl. f. Augenheilk.*, xv., 2, p. 289, and *Therapeutische Monatshefte*, Dec., 1902.

496. **Salzmann.** The atrophy of the choroid in myopic eyes. *Graefe's Archiv*, liv., 2, p. 337.

497. **Widmark.** A contribution to the etiology of myopia. *Hygieia*, Aug.-Sept., 1902.

498. **Jackson, E.** The full correction of myopia. *Ophth. Record*, Nov., 1902.

EINTHOVEN (494) gives, besides a detailed résumé of the literature, a sketch of the accommodation theories of Tscherning-Schoen and Helmholtz, with particular reference to the tremulousness of

the lens in accommodation, discovered by Hess. The writer accepts the Helmholtz theory.

According to LIEBREICH (495) only the nasal portion of the orbit need be taken into consideration in errors of refraction and strabismus, since the centre of rotation of the ball and the ocular muscles have a constant relation to the inner wall only, while their relation to the unsymmetrical temporal half of the orbit varies greatly. The principal measurement is the pupillary distance, which serves to fix the angle B which the two axes of the muscle funnels make with each other. When the angle B is large, the internal recti in fixation must make a supra-normal effort, and thus, according to Liebreich, there is too great an accommodative effect, with the tendency to bring the object fixated too near. In order to exclude the injurious influence of the angle B and to regulate the conditions of accommodation and convergence, Liebreich in the prophylaxis and treatment of myopia orders prisms in order to diminish convergence, or concave glasses to allow a more distant point of fixation, or a combination of the two glasses.

SALZMANN'S (496) valuable paper includes the anatomical examination of six myopic eyes in which he found diffuse and circumscribed changes in the choroid. The former, found in the entire region of the staphyloma posticum, are characterized by great thinning of the choroid with absence of the larger vessels, while the latter are limited to the inner layers or may involve the entire thickness of the choroid later and show absence of the vessels and of the stroma pigment cells. The lamina vitrea is thinned or exhibits breaks. These are at times closed by a new-formed membrane on the inner lamella of the old membrane. As a result of the absence of the capillary layer, there are changes in the pigment epithelium and destruction of the rod and cones and outer nuclear layer.

The whole process, according to Salzmann, begins in the lamina vitrea, since at many points changes in it alone are found. In the development of staphyloma posticum, the lamina vitrea bulges backward and finally ruptures, after which the choroidal vessels degenerate. Inflammation plays in the pathogenesis of these choroidal atrophies only a secondary rôle.

From the anatomical conditions Salzmann concludes that in treating myopic atrophy of the choroid the tension should be kept down. In this sense he believes that the removal of the lens has

a favorable effect, and the improvement in vision after the operation cannot be explained as a purely optical one.

WIDMARK (497) reports from his practice several cases, with weakness of vision congenital or acquired in early life in one eye, in which myopia had developed in the other eye, which at first had good vision.

In some cases in which the one eye was enucleated in childhood, myopia developed in the other eye. These observations led the writer to collect one hundred cases of anisometropia in which there was at least 2. D difference in the refraction of the two eyes and one eye at least was myopic. He found that in a large number of these cases the vision in the lesser myopic or non-myopic eye was weakened by corneal opacities or astigmatism. This fact cannot be explained by the convergence theory. The probable cause seems to the writer to be the different acuteness of vision in the two eyes. The eye with better vision is, in general, that which is chiefly used in near work and therefore the myopia develops mostly in this eye.

The writer has in recent years determined the location of the near point in myopes by having the patient fixate a small flame and finding the point where the corneal image of the flame passes inward in one eye as binocular fixation was lost. He found by this method that most myopes can fixate up to 4*cm.* Traction on the optic nerve at the posterior pole of the eye can therefore hardly occur at the usual working distance. The writer believes that convergence has as little to do in producing myopia as accommodation does.

The writer generally gives myopes the full correction if accommodation and convergence are good. Yet he does not find that the artificially created emmetropia for those eyes which natural emmetropia has not prevented from becoming myopic offers the protection against the increase of myopia which others have reported to have found. Nor does he see any reason for giving myopes of less than 2. D correcting glasses for near work if there are no asthenopic symptoms. DALÉN.

JACKSON (498) reports the histories of 35 cases of myopia treated with full correction. These, with cases already published, make a series of 62. Since myopia, as a rule, is stationary after twenty years of age, the statistics referring to this class, including 35 eyes, are the most instructive. During periods covering from three to eleven years, 60 per cent. did not become more myopic

after beginning to wear glasses, while in 14 eyes the myopia at some time increased more than 0.25 D, in 4 eyes it diminished more than that amount. In nearly all the cases the myopia was clearly progressive before correcting lenses were given. The author's observations confirm the common opinion that full correction is generally proper in uncomplicated cases. ALLING.

VII.—MUSCLES AND NERVES.

499. **Nicolai.** A new ocular muscle—musculus papillæ optici. *Ann. d'ocul.*, cxxviii., p. 342.

500. **Lenoble** and **Aubineau.** Infantile tremors and congenital nystagmus. *Arch. de Neurol.*, 1902, No. 80.

501. **Korniloff.** Associated paralyses of the ocular muscles. *Wjest. ophth.*, 1902, 4–5.

502. **Haltenhoff.** A case of cephalic tetanus with facial and ocular paralysis; recovery. *Revue méd. de la Suisse romande*, Sept. 20, 1902.

503. **v. Frankl-Hochwort.** A case of acute paralysis of the extrinsic muscles supplied by the third nerve, due to neuritis. *Arbeiten aus dem Neurol. Instit. a. d. Wiener Universität*, ix., 1902.

504. **Ackermann.** Complete unilateral paralysis of the third nerve from basilar hemorrhage, with autopsy. *Klin. Monatsbl. f. Augenheilk.*, xl., 2, p. 306.

505. **Strassburger.** Sluggishness of the pupil with accommodation and convergence. *Neurol. Centralbl.*, 1902, No. 16.

506. **Piltz.** The paradoxical reaction of the pupils and a new observation of contraction of the pupils when the eyes were shaded. *Ibid.*, Nos. 20–22.

507. **Jackson, E.** Heterophoria and the indications it gives for treatment. *Amer. Jour. of Ophth.*, Nov., 1902.

508. **Prince.** Section and exsection of the recti muscles for cosmetic effect in cases of squint inoperable by tenotomy and advancement. *Amer. Jour. of Ophth.*, Sept., 1902.

509. **Posey.** Associated movements of the head and eyes. *Jour. Amer. Med. Assoc.*, Nov. 29, 1902.

NICOLAI (499) believes that the van Giesen stain and the trypsin reaction indicate that the fibres lying about the optic disc are unstriped muscle fibres. He distinguishes three groups of fibres in his musculus papillæ optici: (*a*) circular, (*b*) longitudinal, and (*c*) radial fibres. No theory of the physiological significance of the muscle is offered. BERGER.

LENOBLE and AUBINEAU (500) studied the existence of nystagmus without eye disease in its relations to other tremors. They distinguish the following forms:

1. Essential nystagmus without accompanying symptoms.

2. Nystagmus with the existence of independent symptoms, such as inequality of the pupils and asymmetry of the face.

3. Nystagmus with analogous nervous symptoms, such as epileptoid twitchings and increased reflexes.

4. Nystagmus as a symptom of tumor, developing alone or with twitching in other parts of the body.

KORNILOFF (501) reports two cases:

1. In a child of six there developed, after a slight trauma, in the course of two weeks and a half, abnormal eye movements, then headache, vomiting, weakness in the feet and hands, ataxia, normal temperature, slight paresis of the seventh nerve. The lateral and medial movements of the eye are normal, but there is absolute inability to move the eyes up or down. All these symptoms passed off gradually, first in the hands and feet, then the motility of the eye returned, and in six weeks there was complete recovery.

2. A child of four. With a slight increase in temperature the following complexus of symptoms developed: weakness of the feet and of the right hand, disturbances of speech, apathy with preservation of intelligence, limitation of motility in the right hand and right foot, the face somewhat oblique to the right, pronounced Babinsky's symptom, bilateral ptosis, left eye in repose slightly convergent. Motility of both eyes to the right normal, to the left restricted, upward almost abolished.

In the first case a diagnosis of poliencephalitis was made, in the second tubercle in the corpora quadrigemina. The author believes in the existence of supranuclear centres of co-ordination, in favor of which speak cases in which there is inability to move the eyes in some direction by an effort of the will, yet with preserved reflex motility. HIRSCHMANN.

HALTENHOFF (502) describes a very remarkable case of Rose's tetanus with paralysis of the facial and abducens. In a child of three an injury led to prolapse of the lachrymal gland, and this was extirpated. After the operation the wound suppurated, which was attributed to the presence of dirt in the wound. Having in mind the possibility of tetanus infection, Professor Massol advised a prophylactic injection of tetanus serum. Nevertheless, two days later there developed spasms of the gustatory muscles, facial paralysis, and paresis of the abducens upon the injured side. These symptoms passed off after removal of a splinter of wood, and the case ended in complete recovery. This favorable result

was attributed to the use of tetanus serum. It is well known that the cases in which tetanus appears soon after the injury, or is complicated with facial paralysis, are severe; in this case the presence of tetanus germs in the vascular tissue of the orbit might be regarded as rendering the prognosis more grave. In fact the mortality of Rose's tetanus is very great. Of 64 cases which Haltenhoff found in the literature, 39 were fatal. BERGER.

V. FRANKL-HOCHWORT'S (503) case was in a right hemiplegic of sixty-seven, who suddenly was attacked with paralysis of the extrinsic muscles supplied by the right third nerve. The pupils remained normal on each side. The autopsy revealed extensive atheroma but a normal condition of the nuclei of the ocular muscles and of the ciliary ganglia.

The trunk of the right oculomotor exhibited the picture of an acute interstitial neuritis. There was, therefore, a partial oculomotor paralysis due to a localized, non-syphilitic neuritis—a fresh proof that, contrary to Mauthner's dictum, a unilateral paralysis of the extrinsic muscles does not necessarily imply a nuclear lesion.

ACKERMANN'S (504) patient, a boy of fifteen, had a complete left oculomotor paralysis, excepting the sphincter iridis and muscle of accommodation, which were merely paretic. The patient died in coma, and the autopsy showed a hemorrhage above the pons between the arachnoid and the pia.

The oculomotor trunk was surrounded before its exit through the dura by a connective-tissue membrane organized from a blood clot. Microscopic examination showed at this point atrophy with secondary degeneration of the peripheral fibres. Only in the centre of the medial third of the nerve did Weigert's stain show that the nerve fibres were preserved. Since the intrinsic muscles were not completely paralyzed, the writer believes that the fibres which he found normal belonged to these muscles. Their central protected location explains the paralysis of the extrinsic muscles alone.

STRASSBURGER (505) observed in three patients with unilateral or bilateral reflex iridoplegia that the contraction with convergence was well-marked but very sluggish, and the dilatation following contraction required 10–20 seconds instead of 2–3.

PILTZ (506), after a critical examination of the literature, comes to the conclusion that the paradoxical reaction of the pupil

(dilatation on exposure to light) is a very rare condition, and that only four of the cases reported are above criticism. He adds a fifth case of his own, with atrophy of the optic nerves, in which, after illuminating the eyes, the pupils contracted when they were shaded.

JACKSON'S (507) conservative view of the treatment of heterophoria is indicated by the fact that in 714 cases of one-half centrad and upwards only three came to operation. Heterophoria does not necessarily require special treatment since correction of ametropia is generally sufficient. Before resorting to operation, graduated exercises, gymnastics, and general treatment should positively prove to be unavailing. ALLING.

PRINCE (508) has frequently performed exsection of the opposing muscle in cases of paralysis and extreme over-effect of long standing, following extensive tenotomies. One obvious objection to the procedure is the danger of exophthalmus, but it seems not to have proved serious. ALLING.

To six reported cases of torticollis secondary to paralysis of one of the eye muscles, POSEY (509) adds the notes of another which presented the following conditions: A boy of nine had had a wry-neck since infancy, the head drooping upon the right shoulder while the face was turned to the left. The eyes are level in this position, but when the head is straightened the right eye is turned down and the patient fixates with the left. The right superior rectus is probably at fault. Operation was refused and prisms failed to benefit the deformity. Another case of spasmus nictitans is given, in which the child habitually held the head turned to the left side and slightly downward and rotated it from side to side. These movements were rhythmical but under the control of the will. The eyes showed pronounced convergent strabismus of the left with vertical and slightly rotary nystagmus of both. The latter movements were about five times as frequent as those of the head. ALLING.

Sections VIII.–XII. Reviewed by DR. R. SCHWEIGGER, Berlin.

VIII.—LIDS.

510. **Winselmann.** Is the blepharitis characterized by ulceration of the lid margin to be regarded as an eczema? A contribution to the etiology and therapy of blepharitis ulcerosa. *Klin. Monatsbl. f. Augenheilk.*, xlii., 2, p. 393.

511. **Schillinger.** A further case of gangrene of the lid with the presence of diphtheria bacilli. *Inaug. Dissert.*, Tübingen, 1903.

512. **Lotin.** Premature grayness of the lashes and brows. *Wjest. ophth.*, 1902, 4–5.

513. **Panas.** So-called congenital ptosis. *Arch. d'opht.*, xxii., No. 11, p. 677 (with illustrations).

After examining epilated lashes, WINSELMANN (510) concludes that ulcerative blepharitis is a primary parasitic disease of the roots of the lashes similar to sycosis and not to eczema. A secondary blepharitis can of course arise from an eczema. The rational therapy consists in total epilation. Normally there are two sorts of lashes whose relative number differs greatly in blepharitic processes.

SCHILLINGER (511) considers an early bacteriological examination necessary in all forms of spontaneous gangrene of the skin. If positive or even suspicious results are obtained, serum therapy is to be at once resorted to.

LOTIN'S (512) patient was a girl of seventeen in whom in the course of a year, after frequent attacks of migraine, the lashes of the left eye all became gray, and also the medial half of the left and the lateral half of the right brow. The writer believes this grayness to be due to the activity, from some unknown cause, of the pigmentophages described by Metschnikoff. HIRSCHMANN.

PANAS (513) modified his ptosis operation by excising the skin folds lateral to the upper incision in the brow and increased its cosmetic effect. He also operated in Motais's manner, utilizing a portion of the superior rectus tendon, but without any result whatsoever, as well as producing an insufficiency of the muscle and diplopia, which, according to Motais, passes off in two months, but here persisted six months, and necessitated the loosening of the tendon. Then he relieved the ptosis by means of his own operation. V. MITTELSTÄDT.

IX.—LACHRYMAL APPARATUS.

514. **de Lapersonne.** Syphilitic dacryoadenitis. *Arch. d'opht.*, xxii., 12, p. 760.

515. **Lundsgaard.** Do the lachrymal glands atrophy after extirpation of the sac? *Hosp. Tid.*, July, 1902.

516. **Berger.** On epiphora as an initial symptom of Basedow's disease. *Arch. f. Augenheilk.*, xlvi., 2, p. 113.

517. **Panas.** Syphilis of the lachrymal passages. *Arch. d'opht.*, xxii., 12, p. 749.

518. **Adolph.** On extirpation of the lachrymal sac. *Zeitschr. f. Augenheilk.*, viii., 4, p. 441.

De Lapersonne (514) observed in an alcoholic patient of forty, who denied syphilis, a quickly developing bilateral inflammation of the lachrymal glands. Associated with this there was a tedious but painless irido-choroiditis in both eyes and inflammation of the pubic, chest, and salivary glands. Under anti-syphilitic treatment the inflammation of the lachrymal glands gradually receded, while the irido-choroiditis scarcely improved in six weeks. Finally the latter improved and the affection of the glands was reduced to an induration. The writer believed the process to be syphilitic, and thinks that syphilitic affections of the lachrymal glands are not so rare as is commonly supposed. He adds another case in which, following a chancre of the upper lid, the lachrymal sac became involved as a secondary symptom. v. Mittelstädt.

Berger (516) presents several observations of epiphora which seemed to precede Basedow's disease by several years. He believes the cause to be a vasomotor neurosis of the sympathetic which supplies the sac, analogous to the other secretory neuroses found in Basedow's disease—hypersecretion as well as the diminution of lachrymal secretion often observed. There is nothing characteristic about the epiphora and it can be improved only by treatment of the general condition; an extirpation of the gland is contra-indicated because of the probability that the secretion will be much reduced later.

Panas (517) reported a very unusual case of gumma of the lachrymal sac. The patient, aged forty-five, alcoholic, acquired syphilis at the age of twenty and suffered from ozæna with occasional swelling of the region of the left lachrymal sac. The great fungous ulcer and the coexisting tertiary ulcers of the hypertrophied tonsils were cured by inunctions and hypodermic injections of an oily solution of biniodide of mercury. Panas advised against the simultaneous use of iodide of potassium in such cases. In the literature he found but three analogous cases, and he discussed the differential diagnosis between gumma, tuberculosis, and malignant tumors of the sac. v. Mittelstädt.

Adolph (518) records five hundred and sixty-six cases of extirpation of the sac from Kuhnt's clinic, 4.8 % of which healed by second intention. He prefers Kuhnt's technique and regards later modifications as being of little importance. Axenfeld's

speculum does not fulfil its purpose of checking the flow of blood and it tears the tissues.

X.—ORBIT AND NEIGHBORING CAVITIES.

519. **Vossius.** Two rare cases of orbital affection. *30th Heidelberg Ophth. Soc. Report*, 1902. Bergmann, Wiesbaden.

520. **Lindner.** On Krönlein's operation. *Inaug. Dissert.*, 1902, Giessen.

521. **Panas.** Oily cysts in the orbital region. *Arch. d'opht.*, xxii., 12, p. 741.

522. **de Lapersonne.** Orbital and ocular complications of inflammation of the sinuses. *Bull. et mém. d. l. soc. franç. d'opht.*, 1902, 1.

523. **Axenfeld.** A contribution to the pathology and therapy of inflammations of the frontal and ethmoidal sinuses and their orbital complications. *Deutsche med. Wochenschr.*, 1902, No. 45, p. 713.

524. **Bull, C. S.** Cellulitis of the orbit, eyelids, forehead, and cheek following an infected sore of the finger. Treatment by free incisions and subcutaneous injections of sublimate. *Med. News*, Dec. 6, 1902.

Vossius (519) discusses cases of orbital phlegmon in which at the autopsy abscesses were found in the ocular muscles, which partly explain the disturbances of motility. He records also a case of necrosis of the ball from orbital phlegmon, in which after various ocular and general affections had existed for years the autopsy revealed extensive actinomycosis.

Lindner (520) considers Kronlein's operation superior to Knapp's because of the larger field of operation in the orbit secured, so that by its means, for example, even the ciliary ganglion can be removed in glaucoma. It is therefore indicated in orbital phlegmon and in infected wounds, while in tumors of suspected malignancy the greater inspection that it permits is an advantage. The immediate injuries, such as ptosis and the limitation of motility of the ball, improve or disappear in the course of time. It is not so severe that it need be avoided for diagnostic purposes, as two new cases of Vossius show—orbital tumor having been diagnosed and normal orbits found.

Panas (521) removed from the upper-inner margin of the orbit in a girl of ten an oily cyst as large as a pigeon's egg, which had been noticed in the first year of life, but recently had grown rapidly. He found as in three other cases that the superficial wall was composed of connective tissue free from epithelium, while the deep periosteal wall presented a dermoid structure with numerous subaceous glands, a condition explainable by the action on the anterior wall of the growth of the tumor, if one does not

believe that the cyst has arisen from a detached bit of skin included within the tissues. The writer discussed also the differential diagnosis and the treatment. v. MITTELSTÄDT.

The valuable paper by DE LAPERSONNE (522) is not adapted to brief abstracting. He discusses his personal experiences of the symptoms, interesting to ophthalmologists and rhinologists, in diseases of the cavities neighboring the orbit and the various operative methods of treating sinus diseases. BERGER.

A frontal sinus chronically inflamed and causing orbital inflammation should always be chiselled out and radically operated upon, but AXENFELD (523) believes that acute processes do not always require operation, since they may heal spontaneously, while the orbital suppuration, having no outlet, may extend farther. He recommends exploratory trephining as far away as possible from the orbital abscess.

XI.—CONJUNCTIVA.

524 A. **Sprenker.** On the relation of scrophulosis to the common conjunctival and corneal diseases of childhood. *Inaug. Dissert.*, Giessen, 1902.

525. **Demicheri.** Spring catarrh. *Arch. d'opht.*, xxii., No. 10, p. 634.

526. **Terson.** Remarks on conjunctival vegetations recurring in the spring, their structure and their treatment. *Ann. d'ocul.*, cxxviii., p. 330.

527. **Armaignac.** On a case of congenital purulent ophthalmia. *Ibid.*, p. 241.

528. **Fries.** On the treatment of blennorrhœa by Kalt's method. *Inaug. Dissert.*, Tübingen, 1902.

529. **Raehlmann.** On the involvement of the tarsus and lid margin in trachoma. *Arch. f. Augenheilk.*, xlvi., 3, p. 363.

530. **Jacovides.** Trachoma and purulent ophthalmia in Egypt. *Arch. d'opht.*, xxii., 12, p. 767.

531. **Holmström.** Cases of pemphigus of the conjunctiva. *Hygeia*, July, 1902.

532. **Hoppe.** The extension of the retrotarsal conjunctiva upon the upper tarsus. *Monatsbl. f. Augenheilk.*, xl., ii., p. 233.

SPRENKER (524 A) finds from the histories in the Giessen clinic that in only 47 % of the so-called scrofulous ocular affections were there evidences of scrofula, swelling of the glands, eczema, and rhinitis; pronounced tuberculous affections were found in but 2 %. The cornea was more frequently affected (75 %).

DEMICHERI (525) observed spring catarrh in 0.34 % of his cases, chiefly in the form of a disease of the tarsal conjunctiva upon which there were true papillomata. The disease has no relation to

trachoma, which is prevalent in Montevideo. He recommends rubbing the hypertrophied papillæ with calomel, the use of a salve containing white precipitate and cocaine, scraping off the papillæ, and electrolysis, the last although a tedious procedure quickly improving the subjective symptoms. In one case, after removing the papillæ and cauterizing the base, there was a recurrence. (The reviewer has had the same experience.)

v. MITTELSTÄDT.

TERSON (526) finds by anatomical examination that spring catarrh consists chiefly in a hypertrophy of the papillæ. In many cases a relation between spring catarrh and eczema seems probable. By way of treatment Terson recommends yellow oxide of mercury, $\frac{1}{40} - \frac{1}{20}$, in lanolin, and for the secretion protargol, cocaine, adrenalin, and as general measures arsenic and life in high altitudes.

BERGER.

ARMAIGNAC (527) reports a case of congenital blennorrhœa ending in bilateral blindness. Armaignac believes with Kraus of Vienna that gonococcus infection of the vagina may extend into the cavity of the uterus, and that the gonococci pass through the fœtal membranes and enter the amnionic fluid. BERGER.

FRIES (528) confirms the favorable results of Kalt's irrigation method in the blennorrhœa of infants and adults even when there are complicating corneal affections. Since this treatment can be carried out without difficulty and without injury it may be left in the hands of the uninstructed.

RAEHLMANN (529) believes that the grooved curve in the tarsus in trachoma is due to the fact that the consistency of the tarsus itself is altered in trachoma. He believes the beginning of the trachomatous tarsitis to lie in an interstitial blepharitis with swelling about the Meibomian glands and ducts, altering the secretion. He concludes that the tarsus becomes involved at the same time that follicles appear on the tarsal conjunctiva. When cicatricial contraction of the conjunctiva occurs, the softened tarsus curves.

In the portion of his paper that has appeared JACOVIDES (530) combats the idea that the trachoma found in Egypt is distinguished by its severer course from European trachoma. The difference consists only in the fact that of every 10 cases of eye-disease 6–7 are trachomatous. The disease comes on insidiously and therefore is not followed from the beginning unless,

like the writer, one examines at intervals the eyes of healthy persons living with the trachomatous. From the histories of two foreign and two native patients it appears that first a hyperæmia of the tarsal conjunctiva comes on at the outer angle, which extends over into the retrotarsal fold and leads to a thickening of the tissues in which the granulations develop. Variations of this type, such as papillary and mixed trachoma, are found here as in Europe. The latter are found particularly in children from two to twelve years of age. As elsewhere the lower classes are chiefly affected. The prevalent humidity has a very unfavorable influence on the subjective symptoms. v. MITTELSTÄDT.

HOLMSTRÖM (531) describes four cases observed by him of essential shrinking of the conjunctiva. Case 1.—In a woman of forty-five the conjunctival sac gradually cicatrized without visible formation of vesicles. Treatment with arsenic was of no avail. Final result: xerophthalmus. Vesicles were not observed on other parts of the body. Case 2.—A similar cicatrization was gradually developed in a woman of twenty-two. At the same time pemphigus vesicles appeared on the mucosa of the mouth. Treatment with arsenic seemed in this case to be of some efficacy inasmuch as the ocular affection had made no progress in the last six months. The cornea at the time of publication was practically uninvolved. The patient was under observation over a year. Case 3.—A man of fifty. In this patient the ocular affection was accompanied by the formation of pemphigus vesicles in the mouth. Treatment with arsenic brought the ocular affection to a temporary stop. At the time of publication the conjunctivæ were almost obliterated, the corneas clear. The patient was under observation about a year. Case 4.—A woman of twenty-four had a completely developed xerophthalmus of five years' duration. DALÉN.

HOPPE (532) does not agree with Straub that the cicatricial tarsal mucosa in cases of cured trachoma has been drawn over from the retrotarsal folds, but he believes that it is due to loosening of the mucosa from the tarsus and traction upon it. In a case in which no trachoma existed, Hoppe saw, without any inflammatory symptoms, but with the formation of concrements and connective tissue, a diffuse progressive sclerosis of part of the upper palpebral conjunctiva, drawing over the free conjunctiva of the lid onto the tarsus. The process is somewhat similar to the formation of pterygium.

XII.—CORNEA.

533. **Reiss.** On cystic formations on the surface of the cornea. *Klin. Monatsbl. f. Augenheilk.*, xlii., p. 227.

534. **Gallemaerts.** The treatment of keratitis by electrolysis. Brussels, 1901.

535. **Weinstein.** On the healing of incised wounds of the cornea. *Wjest. ophth.*, 1902, 4–5.

536. **Suker.** A fistula of the cornea with a hernia of the iris and a cyst of the conjunctiva connected with the anterior chamber. *Ophth. Record*, Sept., 1902.

Reiss (533) found in a blind eye of normal size the entire corneal region filled with cysts that lay before the cornea and were formed of conjunctiva that had been drawn over it. This had followed ophthalmia neonatorum fourteen years before. Furthermore he found chalk in the scar, bone in the choroid, and thickening of the lamina vitrea.

Gallemaerts (534) uses electrolysis in pannus, ribbon-shaped keratitis, and scrofulous corneal ulcers. The negative pole is used with a current of 2 m.a. This procedure causes but little irritation and improves quickly the old cases, soon causing a cessation of the photophobia.

Weinstein's (535) investigations led to the following results: 1. An hour after the cornea had been incised the wound contained only a plug of fibrin; immediate contact between the central lamellæ of the substantia propria was observed only in one small wound among 200 cases. 2. Four hours after the injury the epithelium is clearly seen dipping into the wound. Twenty-four to forty-eight hours later the entire anterior portion of the wound is filled with epithelium. 3. This indipping is due to an active process of proliferation shown by the existence of kariokinetic figures in the epithelial cells. 4. This process reaches its highest point in four hours. 5. There is no regularity in the distribution of the mitoses on the surface of the cornea. 6. In the epithelium which fills the wound the most varied kariokinetic figures are seen four hours after the operation. 7. The kariokinetic process diminishes toward the end of the first twenty-four hours but does not cease entirely. 8. There is always a superfluity of the epithelium filling the wound. 9. As the regeneration of the substantia propria proceeds under the epithelium the epithelial plug is forced out. 10. The epithelium in the region of the wound never returns to the normal but is two to

four times as thick and fills up the space left by the retraction of the scar.

In regard to Descemet's membrane, the writer differs from all others in believing that it is replaced after penetrating wounds, becoming in four months as thick as before. HIRSCHMANN.

SUKER (536) describes a cyst of the sclero-corneal margin 10*mm* long, 5*mm* broad, and 5*mm* deep, which was connected with the anterior chamber and contained a knuckle of iris. The cyst was laid open and the base scraped, after which there was no recurrence. ALLING.

BOOK NOTICES.

XVI. — **Hermann von Helmholtz,** von LEO KÖNIGSBERGER. Vieweg & Sohn: Braunschweig, 1902. In three beautifully printed 8vo volumes with 9 heliogravures and a facsimile of a letter.

This excellent biography is worthy of the great scientist. The author gives an extensive history of the life and achievements of Helmholtz, which the reviewer can only sketch in this place, expecting to furnish for the January number of these ARCHIVES a condensed but detailed account of Helmholtz's work, based on the admirable volumes of Professor Königsberger of Heidelberg.

Helmholtz's father was a professor at the Lyceum of Potsdam (Prussia), teaching Latin and Greek classics, and mathematics. His mind had a turn toward philosophy, with devotion to Kant and Fichte. Helmholtz, born Aug. 31, 1827, was not remarkable as a child, but from his eighth year his progress at school was equal to the better scholars of his class, with the addition of a bias and aptitude for architectural and geometrical constructions. In the Lyceum he took up all the prescribed studies of the regular course, and some optional ones, among which was Hebrew. In his eighteenth year he passed the examination of maturity, receiving a "very good" certificate. His love of mathematics and natural science induced him to ask his father whether he could not make these studies his life work. His father replied: "You can, if you include medicine." Accepting the proposition, he entered the Military Medical School of Berlin, where he graduated with honor. In his inaugural dissertation ("De Fabrica Systematis Nervosi Evertebratorum), November 2, 1842, he described an important microscopical discovery, namely, the connection of nerve fibres with ganglion cells, by which the latter were recognized as nerve-cells. From now there followed an uninterrupted series of discoveries and inventions: the nature of putrefaction and fermentation, 1843; after this a number of

papers to demonstrate the great law of the universe, the *preservation of force* (the constance of energy), which he proved for the organic nature as well as for the inorganic (1849 and 1850); the *invention of the ophthalmoscope* was in 1851. In the year 1849 he was appointed professor of physiology at Königsberg, where he determined, in 1850, the *velocity of the nerve-current*, by means of an instrument to *measure smallest intervals of time*, and he made other more extended experiments on kindred subjects; in 1852, on the *theory of compound colors*. In 1855 there appeared his celebrated paper on the "Accommodation of the Eye," in *Graefe's Archiv*, and the first part of his *great treatise on Physiological Optics*.

In 1855 he was appointed professor of anatomy and physiology in Bonn: in 1858 professor of physiology in Heidelberg, where he remained until 1871, to follow a call to Berlin as professor of physics, where he and his friend Dubois-Reymond divided themselves into the new large physico-physiological Institute. In Heidelberg he founded in conjunction with Bunsen and Kirchhoff, "a period of splendor as it rarely existed in any university, and not easily will be seen again." There he wrote the greater part of his large *Handbuch der Physiologischen Optik*, the first part of which appeared in 1856, the second in 1860, the third and last in 1866. He carefully went over the whole field, critically re-examining every subject, proving, amending, and correcting any question, then presenting it historically and scientifically, so simply and concisely as clearness would permit. He resorted frequently to the aid of mathematics, but rarely in a higher grade than which is taught in any reputable college. This large and fundamental work was translated into French by Dr. E. Javal, of Paris; it soon was out of print, and a second edition was prepared by Helmholtz himself, assisted by Prof. Arthur König. It has a complete bibliography of every original publication on physiologic optics. The first edition was published as Bd. ix. of Karsten's *Allgemeine Encyclopädie der Physik*, the second by Leopold Voss, Hamburg and Leipzig, 1896. Besides this treatise he wrote another, *Die Lehre von den Tonempfindungen*, 1863. This is a smaller work, but contains the result of most remarkable investigations, especially on the *Clangcolor* (*timbre*) of vowels, which, besides pitch and intensity, is a quality of sound that had not previously been accounted for. A considerable number of other investigations on sound were published as independent

papers and in his *Sensations of Sound.* During these occupations in ophthalmic and aural physiology, he took up new questions of interest in general physics. He made repeated journeys abroad, to France and especially to England. In 1893 he visited America as a delegate to the committee which was commissioned to determine, at the Exposition of Chicago, international units of certain electrical values and energies.

He was married twice, first to Olga von Velten, and then to Anna, daughter of Professor, and later Minister, Rob. v. Mohl. He died in 1894, survived by a son of his first wife, a manufacturing engineer, and two sons (who are dead), and a daughter of his second wife, who is married to a son of the celebrated electrician and naturalist, Werner von Siemens, of Berlin. She has several children.

In concluding these notes, we desire to say that the Helmholtz-Biography by L. Königsberger is a classical work, elegant and fascinating in style, as instructive as it is attractive, portraying a remarkable period in the progress of science and civilization, by the insight we gain into the personality, character, and working habits not only of one of the foremost naturalists but of many of the keenest minds of his time, his co-operators, with whom he was in constant touch by friendly and literary intercourse.

H. KNAPP.

XVII.—**A Handbook of the Diseases of the Eye and their Treatment.** By HENRY R. SWANZY, A.M., M.B., F.R.C.S.I. Eighth edition, with 168 illustrations, 565 pages in 12mo, with a good index. Price, $2.50. Philadelphia: P. Blackiston's Son & Co., 1903.

A text-book at the eighth edition requires no recommendation, if the author takes care not to lag gradually in the race. This new edition has received many additions, viz.: Conjunctivitis Petrificans, Grating Keratitis, Keratitis Aspergillina, Pflüger's Method of Tarsorraphy, more detailed accounts of Sympathetic Ophthalmitis, of Kuhnt's Method of Extirpating the Lachrymal Sac, the Use of the Magnet for Foreign Bodies in the Eye, Tumors of the Optic Nerve, Krönlein's Temporary Resection of the Outer Wall of the Orbit, a description of Amaurotic Family Idiocy, and others. Chapter II. (33 pages) — Refraction and Accommodation — is insufficient for an ophthalmic practitioner. The admission of kindred forms of corneal opacities of a mild chronic character, which have received attention during the last

few years only, into text-books is seasonable and praiseworthy,— we mean the Grating Keratitis of Haab, the Guttate Keratitis of Groenouw, the Superficial Punctate Keratitis of Fuchs, and the Sclerosing Opacity of the Cornea, all of which have more or less the same character — dimness of sight, irregular punctate or striated opacities in a somewhat diffuse corneal haze. They are not objects of daily practice, yet sufficiently frequent that every experienced eye surgeon, in reading the descriptions, is reminded of such cases in his practice. The final clearing of the cornea, in the experience of the reviewer, is not so frequent as the descriptions would lead one to believe. The lack of efficient treatment is sorrowfully felt by patient and physician. These forms, if they damage sight more or less, terminate in blindness only exceptionally.

Fig. 60, p. 196, gives an excellent illustration of A. Critchett's case of Solid Œdema, or Elephantiasis Lymphangiodes of the Eyelids, taken from the *Trans. Ophth. Soc. U. K.*, vol. xix. There is, in this affection, almost invariably, a history of recurring facial erysipelas. Operative and other treatment yield no permanent cures. The extirpation of the lachrymal sac is carefully described. Sympathetic ophthalmia and the motions of the pupil in health and disease receive perhaps more space than their share. The diagnosis and removal of iron foreign bodies are well presented and illustrated. The ocular muscles are duly discussed.

The book being handy and well printed, warrants a good prognosis of coming out in many more editions. H. K.

ANNOUNCEMENT.

We copy the following details from the *British Medical Journal*, September 12, 1903, in addition to our short announcement of the next International Ophthalmological Congress, p. 515 this volume.

Professor Dufour is President, Professor Pflüger, Vice-President, Professor Mellinger, Secretary and Treasurer of the Organizing Committee, Professor Snellen of Utrecht, President of the last Congress, is an honorary President. The Organizing Committee has issued a kind of manifesto, certain passages of which we think might well be borne in mind by all that take an active part in the organization of congresses. The Committee declares that scientific congresses have been reproached, and with reason, for failing in their aim—the advancement and diffusion of science.

The reasons for this are, first, that too much time is given up to excursions and pleasure. In the second place, the rapid publication of scientific journals renders superfluous a long journey to hear communications which could be better understood by reading. The Committee rightly insists that discussion, not the mere reading of papers, should be the chief object of these meetings. "By interchange of ideas the spark is generated that brings us new light." With the object of rendering the discussions more profitable, the Organizing Committee has made the following arrangements: Members who wish to contribute papers are requested to send their manuscripts at the same time as their subscription to Prof. Mellinger, Bâle, before May 1, 1904. The paper must be written in one of the four official languages of the Congress: English, French, German, Italian, and its length must not exceed five printed pages of the size of the official reports of the previous congresses. The papers received will be grouped according to their contents and immediately printed. They will form the first part of the printed report of the Congress, and will be sent to each member, together with his admission ticket, at least two weeks before the time appointed for the opening of the Congress. In this way each member will know the subjects to be dealt with, and will be able to prepare himself for serious discussion. The Committee suggests that by this means the reading of the papers may be dispensed with—a notable reform which may save congresses from the fate which seems otherwise to be in store for them by crumbling away under their own weight. The authors will be called upon by the President to make known in a few words the conclusions at which they have arrived, after which the discussion will at once begin. It is hoped that in this way the opinions of experienced men, who have not the time to write and publish their experiences and opinions, may be heard. The discussions will be printed and will form the second part of the official report, which will be sent to each member after the close of the Congress. Only one subject is officially proposed for discussion, namely, "To settle the question of indemnity as regards the value of an eye, lost or injured." Professor Axenfeld (Freiburg), Dr. Sulzer (Paris), and Dr. Würdemann (Milwaukee) have been invited to draw up reports on the subject, which will be printed at the beginning of the official report.

The afternoons will be given up to practical demonstrations.

Notwithstanding the somewhat austere sentiments professed by

the Committee, "cakes and ale" will not be altogether neglected. The town of Lucerne proposes to give a reception on the eve of the opening of the Congress, September 18th. On September 20th there will be an official dinner. Mountain or lake excursions are also proposed. The entrance fee, 25 fr., will entitle members to receive the official report of the Congress and to take part in the excursions.

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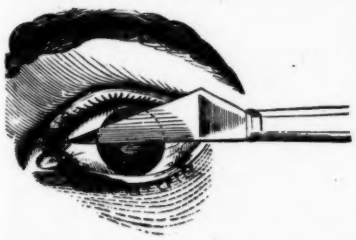

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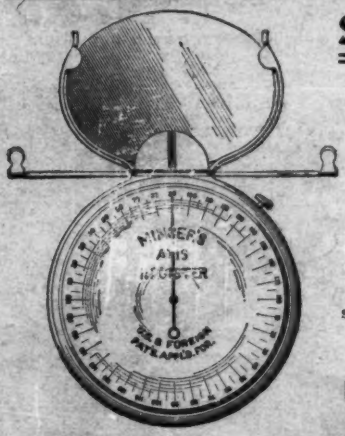

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